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YOU HAVE REQUESTED DATA FROM 34 ANSWERS - CONTINUE? Y/(N):y

L3 ANSWER 1 OF 34 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1998:303296 BIOSIS
DN PREV199800303296
TI Isolation of new Arabidopsis mutants with enhanced disease susceptibility to Pseudomonas syringae by direct screening.
AU Volko, Sigrid M.; Boller, Thomas; ***Ausubel, Frederick M. (1)***
CS (1) Dep. Mol. Biol., Mass. Gen. Hosp., Boston, MA 02114 USA
SO Genetics, (June, 1998) Vol. 149, No. 2, pp. 537-548.
ISSN: 0016-6731.
DT Article
LA English
AB To identify plant defense components that are important in restricting the growth of virulent ***pathogens*** , we screened for Arabidopsis mutants in the accession Columbia (carrying the transgene BGL2-GUS) that display enhanced disease susceptibility to the virulent bacterial ***pathogen*** Pseudomonas syringaepv.

maculicola (Psm) ES4326. Among six (out of a total of II isolated) enhanced disease susceptibility (eds) mutants that were studied in detail, we identified one allele of the previously described npr1/nim1/sail mutation, which is affected in mounting a systemic acquired resistance response, one allele of the previously identified EDS5 gene, and four EDS genes that have not been previously described. The six eds mutants studied in detail (npr1-4, eds5-Z eds10-1, eds11-1, eds12-1, and eds13-1) displayed different patterns of enhanced susceptibility to a variety of phytopathogenic bacteria and to the obligate biotrophic fungal ***pathogenErysiphe*** orontii suggesting that particular EDS genes have ***pathogen*** -specific roles in conferring resistance. All six eds mutants retained the ability to mount a hypersensitive response and to restrict the growth of the avirulent strain Psm ES4326/avrRpt2. With the exception of npr1-4, the mutants were able to initiate a systemic acquired resistance (SAR) response, although enhanced growth of Psm ES4326 was still detectable in leaves of SAR-induced plants. The data presented here indicate that eds genes define a variety of components involved in limiting ***pathogen*** growth, that many additional EDS genes remain to be discovered, and that direct screens for mutants with altered susceptibility to ***pathogens*** are helpful in the dissection of complex ***pathogen*** response pathways in plants.

L3 ANSWER 2 OF 34 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1998:271836 BIOSIS
DN PREV199800271836
TI A light-independent developmental mechanism potentiates flavonoid gene expression in Arabidopsis seedlings.
AU Kubasek, William L.; ***Ausubel, Frederick M.*** ; Shirley, Brenda Winkel (1)
CS (1) Dep. Biol., Virginia Polytechnic Inst., Blacksburg, VA 24061-0406 USA
SO Plant Molecular Biology, (May, 1998) Vol. 37, No. 2, pp. 217-223. ISSN: 0167-4412.
DT Article
LA English
AB Throughout the plant kingdom expression of the flavonoid biosynthetic pathway-is precisely regulated in response to developmental signals, nutrient status, and environmental stimuli such as light, heat and ***pathogen*** attack. Previously we showed that, in developing Arabidopsis seedlings, flavonoid genes are transiently expressed during germination in a light-dependent manner, with maximal mRNA levels occurring in 3-day-old seedlings. Here we describe the relationship between developmental and

environmental regulation of flavonoid biosynthesis by examining phenylalanine ammonia-lyase (PAL), chalcone synthase (CHS), chalcone isomerase (CHI), and dihydroflavonol reductase (DFR) mRNA levels in germinating Arabidopsis seedlings as a function of light, developmental stage and temperature. We show that seedlings exhibit a transient potential for induction of these four genes, which is distinct from that observed for chlorophyll a/b-binding protein(CAB). The potential for flavonoid gene induction was similar in seedlings crown in darkness and red light, indicating that induction potential is not linked to cotyledon expansion or the development of photosynthetic capacity. The evidence for metabolic regulation of flavonoid genes during seedling development is discussed.

L3 ANSWER 3 OF 34 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1998:99683 BIOSIS
DN PREV199800099683
TI The epidemiology of resistance to ofloxacin and oxacillin among clinical coagulase-negative staphylococcal isolates: Analysis of risk factors and strain types.
AU Pegues, David A.; Colby, Christine; Hibberd, Patricia L.; Cohen, Louise Glassner; ***Ausubel, Frederick M.*** ; Calderwood, Stephen B.; Hooper, David C. (1)
CS (1) Infectious Disease Division, Massachusetts General Hosp., 55 Fruit St., Boston, MA 02114-2696 USA
SO Clinical Infectious Diseases, (Jan., 1998) Vol. 26, No. 1, pp. 72-79.
ISSN: 1058-4838.
DT Article
LA English
AB Coagulase-negative staphylococci are important nosocomial ***pathogens*** that increasingly are resistant to oxacillin and fluoroquinolones. To determine predictors of acquisition of oxacillin and ofloxacin resistance, we prospectively identified 150 patients from whose clinical specimens coagulase-negative staphylococci were isolated that differed in susceptibility to oxacillin and ofloxacin. In multivariate analysis, isolation of ofloxacin-resistant coagulase-negative staphylococci was associated with receipt of aminoglycosides (odds ratio (OR) = 8.45; 95% confidence interval (CI) = 2.10-34.1; P = .001) and fluoroquinolones (OR = 11.50; 95% CI = 4.15-31.6; P < .001) within 30 days; oxacillin resistance was associated with prior receipt of beta-lactam agents (OR = 5.99; 95% CI = 2.91-12.3; P < .001). Among oxacillin-resistant strains, there was heterogeneity of pulsed-field gel electrophoresis (PFGE) types, and no type was common between ofloxacin-resistant and

ofloxacin-susceptible strains. Thus ofloxacin resistance may have emerged de novo among diverse oxacillin-resistant strains following the selection pressures of antimicrobial therapy. In contrast, 50% of patients with oxacillin-susceptible/ofloxacin-resistant strains had one of two PFGE types, a finding suggesting that person-to-person transmission resulted in the dissemination of some of these strains.

L3 ANSWER 4 OF 34 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1998:52861 BIOSIS
DN PREV199800052861
TI Inactivation of the alpha C protein antigen gene, bca, by a novel shuttle/suicide vector results in attenuation of virulence and immunity in group B Streptococcus.
AU Li, Jing; Kasper, Dennis L.; ***Ausubel, Frederick M.*** ; Rosner, Bernard; Michel, James L. (1)
CS (1) Channing Lab., 181 Longwood Ave., Boston, MA 02115 USA
SO Proceedings of the National Academy of Sciences of the United States of America, (Nov. 25, 1997) Vol. 94, No. 24, pp. 13251-13256. ISSN: 0027-8424.
DT Article
LA English
AB The alpha C protein of group B Streptococcus (GBS) is a major surface-associated antigen. Although its role in the biology and virulence of GBS has not been defined, it is opsonic and capable of eliciting protective immunity. The a C protein is widely distributed among clinical isolates and is a potential protein carrier and antigen in conjugate vaccines to prevent GBS infections. The structural gene for the alpha C protein, bca, has been cloned and sequenced. The protein encoded by bca is related to a class of surface-associated proteins of Gram-positive cocci involved in virulence and immunity. To investigate the potential roles of the alpha C protein, bca null mutants were generated in which the bca gene was replaced with a kanamycin resistance cassette via homologous recombination using a novel shuttle/suicide vector. Studies of lethality in neonatal mice showed that the virulence of the bca null mutants was attenuated 5- to 7-fold when compared with the isogenic wild-type strain A909. Significant differences in mortality occurred in the first 24 h, suggesting that the role of the alpha antigen is important in the initial stages of the infection. In contrast to A909, bca mutants were no longer killed by polymorphonuclear leukocytes in the presence of alpha-specific antibodies in an in vitro opsonophagocytic assay. In contrast to previous studies, alpha antigen expression does not appear to play a role in resistance to opsonophagocytosis in the absence of

alpha-specific antibodies. In addition, antibodies to the alpha C protein did not passively protect neonatal mice from lethal challenge with bca mutants, suggesting that these epitopes are uniquely present within the alpha antigen as expressed from the bca gene. Therefore, the alpha C protein is important in the ***pathogenesis*** of GBS infection and is a target for protective immunity in the development of GBS vaccines.

L3 ANSWER 5 OF 34 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1998:49050 BIOSIS
DN PREV199800049050
TI Use of model plant hosts to identify Pseudomonas aeruginosa virulence factors.
AU Rahme, Laurence G.; Tan, Man-Wah; Le, Long; Wong, Sandy M.; Tompkins, Ronald G.; Calderwood, Stephen B.; ***Ausubel, Frederick*** *** M. (1)***
CS (1) Dep. Genetics Harvard Med. Sch., Dep. Molecular Biol., Massachusetts General Hosp., Boston, MA 02114 USA
SO Proceedings of the National Academy of Sciences of the United States of America, (Nov. 25, 1997) Vol. 94, No. 24, pp. 13245-13250. ISSN: 0027-8424.
DT Article
LA English
AB We used plants as an in vivo ***pathogenesis*** model for the identification of virulence factors of the human opportunistic ***pathogen*** Pseudomonas aeruginosa. Nine of nine TnphoA mutant derivatives of P. aeruginosa strain UCBPPPA14 that were identified in a plant leaf assay for less ***pathogenic*** mutants also exhibited significantly reduced ***pathogenicity*** in a burned mouse ***pathogenicity*** model, suggesting that P. aeruginosa utilizes common strategies to infect both hosts. Seven of these nine mutants contain TnphoA insertions in previously unknown genes. These results demonstrate that an alternative nonvertebrate host of a human bacterial ***pathogen*** can be used in an in vivo high throughput screen to identify novel bacterial virulence factors involved in mammalian ***pathogenesis*** .

L3 ANSWER 6 OF 34 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1997:250031 BIOSIS
DN PREV199799549234
TI Phytoalexin-deficient mutants of arabidopsis reveal that PAD4 encodes a regulatory factor and that four PAD genes contribute to downy mildew resistance.
AU Glazebrook, Jane (1); Zooki, Michael; Mert, Figen; Kagan, Isabelle; Rogers, Elizabeth E.; Crute, Ian R.; Holub, Eric B.; Hammerschmits,

Raymond; ***Ausubel, Fredrick M.***
CS (1) Cent. Agric. Biotechnol., 5128 Plant Science Build., Univ. Maryland, College Park, MD 20742 USA
SO Genetics, (1997) Vol. 146, No. 1, pp. 381-392. ISSN: 0016-6731.
DT Article
LA English
AB We are working to determine the role of the Arabidopsis phytoalexin, camalexin, in protecting the plant from ***pathogen*** attack by isolating phytoalexin-deficient (pad) mutants in the accession Columbia (Col-0) and examining their response to ***pathogens*** . Mutations in PAD1, PAD2, and PAD4 caused enhanced susceptibility to the bacterial ***pathogen*** Pseudomonas syringae pv. maculicola strain ES4326 (PsmES4326), while mutations in PAD3 or PAD5 did not. Camalexin was not detected in any of the double mutants pad1-1 pad2-1, padl-1 pad3-1 or pad2-1 pad3-1. Growth of PsmES4326 in pad1-1 pad2-1 was greater than that in pad1-1 or pad2-1 plants, while growth in padl-1 pad3-1 and pad2-1 pad3-1 plants was similar to that in pad1-1 and pad2-1 plants, respectively. The pad4-1 mutation caused reduced camalexin synthesis in response to PsmES4326 infection, but not in response to Cochliobolus carbonum infection, indicating that PAD4 has a regulatory function. PAD1, PAD2, PAD3 and PAD4 are all required for resistance to the eukaryotic biotroph Peronospora parasitica. The pad4-1 mutation caused the most dramatic change, exhibiting full susceptibility to four of six Col-incompatible parasite isolates. Interestingly, each combination of double mutants between padl-1, pad2-1 and pad3-1 exhibited additive shifts to moderate or full susceptibility to most of the isolates.

L3 ANSWER 7 OF 34 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1997:204114 BIOSIS
DN PREV199799503317
TI Arabidopsis enhanced disease susceptibility mutants exhibit enhanced susceptibility to several bacterial ***pathogens*** and alterations in PR-1 gene expression.
AU Rogers, Elizabeth E.; ***Ausubel, Frederick M. (1)***
CS (1) Dep. Molecular Biol., Massachusetts General Hosp., Boston, MA 02114 USA
SO Plant Cell, (1997) Vol. 9, No. 3, pp. 305-316. ISSN: 1040-4651.
DT Article
LA English
AB To identify plant defense responses that limit ***pathogen*** attack, Arabidopsis eds mutants that exhibit enhanced disease

susceptibility to the virulent bacterial ***pathogen*** Pseudomonas syringae pv maculicola ES4326 were previously identified. In this study, we show that each of four eds mutants (eds5-1, eds6-1, eds7-1, and eds9-1) has a distinguishable phenotype with respect to the degree of susceptibility to a panel of bacterial phytopathogens and the ability to activate ***pathogenesis*** -related PR-1 gene expression after ***pathogen*** attack. None of the four eds mutants exhibited observable defects in mounting a hypersensitive response. Although all four eds mutants were also capable of mounting a systemic acquired resistance response, enhanced growth of P. s. maculicola ES4326 was still apparent in the secondarily infected leaves of three of the eds mutants. These data indicate that eds genes define a diverse set of previously unknown defense responses that affect resistance to virulent ***pathogens*** .

L3 ANSWER 8 OF 34 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1997:71134 BIOSIS
DN PREV199799370337
TI Molecular recognition of ***pathogen*** attack occurs inside of plant cells in plant disease resistance specified by the Arabidopsis genes RPS2 and RPM1.
AU Leister, R. Todd; ***Ausubel, Fredrick M.*** ; Katagiri, Fumiaki (1)
CS (1) Dep. Biol. Sci., Univ. Md. Baltimore County, 1000 Hilltop Circle, Baltimore, MD 21250 USA
SO Proceedings of the National Academy of Sciences of the United States of America, (1996) Vol. 93, No. 26, pp. 15497-15502. ISSN: 0027-8424.
DT Article
LA English
AB The Arabidopsis thaliana disease resistance genes RPS2 and RPM1 belong to a class of plant disease resistance genes that encode proteins that contain an N-terminal tripartite nucleotide binding site (NBS) and a C-terminal tandem array of leucine-rich repeats. RPS2 and RPM1 confer resistance to strains of the bacterial phytopathogen Pseudomonas syringae carrying the avirulence genes avr-Rpt2 and avrB, respectively. In these gene-for-gene relationships, it has been proposed that ***pathogen*** avirulence genes generate specific ligands that are recognized by cognate receptors encoded by the corresponding plant resistance genes. To test this hypothesis, it is crucial to know the site of the potential molecular recognition. Mutational analysis of RPS2 protein and in vitro translation/translocation studies indicated that RPS2 protein is localized in the plant cytoplasm. To determine

whether avirulence gene products themselves are the ligands for resistance proteins, we expressed the avr-Rpt2 and avrB genes directly in plant cells using a novel quantitative transient expression assay, and found that expression of avr-Rpt2 and avrB elicited a resistance response in plants carrying the corresponding resistance genes. This observation indicates that no bacterial factors other than the avirulence gene products are required for the specific resistance response as long as the avirulence gene products are correctly localized. We propose that molecular recognition of P. syringae in RPS2- and RPM1-specified resistance occurs inside of plant cells.

L3 ANSWER 9 OF 34 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1996:542562 BIOSIS
DN PREV199699264918
TI Mode of action of the Arabidopsis thaliana phytoalexin camalexin and its role in Arabidopsis- ***pathogen*** interactions.
AU Rogers, Elizabeth E.; Glazebrook, Jane; ***Ausubel, Frederick M.*** *** (1)***
CS (1) Dep. Genetics, Harvard Med. Sch., Massachusetts Gen. Hosp., Boston, MA 02114 USA
SO Molecular Plant-Microbe Interactions, (1996) Vol. 9, No. 8, pp. 748-757.
ISSN: 0894-0282.
DT Article
LA English
AB The virulent Arabidopsis thaliana ***pathogen*** Pseudomonas syringae pv. maculicola strain ES4326 (Psm ES4326) and other gram-negative bacteria are sensitive to camalexin (3-thiazol-2'-yl-indole), the Arabidopsis phytoalexin. Furthermore, Psm ES4326 is unable to degrade camalexin or to become tolerant to it. Apparently, Psm ES4326 is a successful ***pathogen*** even though it elicits synthesis of a host phytoalexin to which it is sensitive. Assays of membrane integrity revealed that, like other phytoalexins, camalexin disrupts bacterial membranes, suggesting that camalexin toxicity is a consequence of membrane disruption. A screen for camalexin-resistant mutants of Psm ES4326 yielded only partially resistant mutants, which displayed partial resistance in both killing and membrane integrity assays. These mutants were also resistant to low concentrations of tetracycline and nalidixic acid, suggesting that they were affected in components of the outer membrane. The mutants were not distinguishable from Psm ES4326 in virulence assays. Camalexin was toxic to Arabidopsis cells growing in tissue culture. However, comparison of the extent of cell death associated with disease symptoms in infected leaves of wild-type

Arabidopsis and a camalexin-deficient mutant suggested that camalexin does not contribute significantly to cell death in infected tissue.

L3 ANSWER 10 OF 34 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1996:539311 BIOSIS
DN PREV199699261667
TI Use of cleaved amplified polymorphic sequences to distinguish strains of Staphylococcus epidermidis.
AU Calderwood, Stephen B. (1); Baker, Meghan A.; Carroll, Patricia A.; Michel, James L.; Arbeit, Robert D.; ***Ausubel, Frederick M.***
CS (1) Infectious Dis. Unit, Mass. Gen. Hosp., Boston, MA 02114 USA
SO Journal of Clinical Microbiology, (1996) Vol. 34, No. 11, pp. 2860-2865.
ISSN: 0095-1137.
DT Article
LA English
AB We examined the utility of a PCR-based method termed cleaved amplified polymorphic sequences (CAPS) to type 35 well-characterized isolates of Staphylococcus epidermidis. The results were compared with detailed epidemiologic information and typing obtained by using pulsed-field gel electrophoresis (PFGE). To identify CAPS markers for this study, eight pairs of oligonucleotide primers corresponding to five previously sequenced S. epidermidis genes were synthesized and then used to amplify DNA sequences from the S. epidermidis strains by using PCR. Amplified products were reproducibly obtained for seven of eight primer pairs from chromosomal DNA of 33 of the 35 isolates. Seven restriction site polymorphisms were found in five of the amplified products when they were subjected to digestion with a panel of restriction endonucleases. Each fragment-enzyme combination that was polymorphic demonstrated only two alleles in the 33 S. epidermidis isolates analyzed, corresponding to the presence or absence of a single restriction site. Overall, five distinct combinations of alleles were detected and were designated CAPS types A through E. There was a close correlation between the CAPS grouping, the epidemiologic information for the strains, and grouping by PFGE following SmaI digestion of chromosomal DNA. Although PFGE analysis was more discriminatory than typing based on the limited number of CAPS markers used in this study (isolates from the same CAPS group were sometimes distributed into more than one PFGE group), no isolates from the same PFGE group were found in more than one CAPS group. The CAPS procedure was highly reproducible, in contrast to published experience with arbitrarily primed PCR. These preliminary data suggest that CAPS represents a PCR-based technique for strain typing that is highly reproducible, rapid, utilizes

widely available technologies, and provides results that are relatively easy to interpret and express.

L3 ANSWER 11 OF 34 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1996:316859 BIOSIS
DN PREV199699039215
TI Isolation of arabidopsis mutants with enhanced disease susceptibility by direct screening.
AU Glazebrook, Jane (1); Rogers, Elizabeth E.; ***Ausubel, Frederick*** *** M.***
CS (1) Cent. Agric. Biotechnol., 1105 Ag/Life Sci. Surge Building, College Park, MD 20742-3351 USA
SO Genetics, (1996) Vol. 143, No. 2, pp. 973-982. ISSN: 0016-6731.
DT Article
LA English
AB To discover which components of plant defense responses make significant contributions to limiting ***pathogen*** attack, we screened a mutagenized population of Arabidopsis thaliana for individuals that exhibit increased susceptibility to the moderately virulent bacterial ***pathogen*** Pseudomonas syringae pv. maculicola ES4326 (Psm ES4326). The 12 enhanced disease susceptibility (eds) mutants isolated included alleles of two genes involved in phytoalexin biosynthesis (pad2, which had been identified previously, and pad4, which had not been identified previously), two alleles of the previously identified npr1 gene, which affects expression of other defense genes, and alleles of seven previously unidentified genes of unknown function. The npr1 mutations caused greatly reduced expression of the PR1 gene in response to PsmES4326 infection, but had little effect on expression of two other defense genes, BGL2 and PR5, suggesting that PR1 expression may be important for limiting growth of PsmES4326. While direct screens for mutants with quantitative ***pathogen*** -susceptibility phenotypes have not been reported previously, our finding that mutants isolated in this way include those affected in known defense responses supports the notion that this type of screening strategy allows genetic dissection of the roles of various plant defense responses in disease resistance.

L3 ANSWER 12 OF 34 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1996:193670 BIOSIS
DN PREV199698749799
TI Isolation of Arabidopsis genes that differentiate between resistance responses mediated by the RPS2 and RPM1 disease resistance genes.
AU Reuber, T. Lynne; ***Ausubel, Frederick M. (1)***

CS (1) Dep. Mol. Biol., Massachusetts Gen. Hosp., Boston, MA 02114 USA
SO Plant Cell, (1996) Vol. 8, No. 2, pp. 241-249.
ISSN: 1040-4651.
DT Article
LA English
AB The Arabidopsis disease resistance gene RPS2 is involved in recognition of bacterial ***pathogens*** carrying the avirulence gene avrRpt2, and the RPM1 resistance gene is involved in recognition of ***pathogens*** carrying avrRpm1 or avrB. We identified and cloned two Arabidopsis genes, AIG1 and AIG2 (for avrRpt2-induced gene), that exhibit RPS2- and avrRpt2-dependent induction early after infection with Pseudomonas syringae pv macuticola strain ES4326 carrying avrRpt2. However, ES4326 carrying avrRpm1 or avrB did not induce early expression of AIG1 and AIG2. Conversely, ES4326 carrying avrRpm1 or avrB induced early expression of the previously isolated defense-related gene EL13, whereas ES4326 carrying avrRpt2 did not. The induction patterns of the AIG genes and EL13 demonstrate that different resistance gene-avr gene combinations can elicit distinct defense responses. Furthermore, by examining the expression of AIG1 and EL13 in plants infiltrated with a mixed inoculum of ES4326 carrying avrRpt2 and ES4326 carrying avrRpm1, we found that there is interference between the RPS2- and RPM1-mediated resistance responses.

L3 ANSWER 13 OF 34 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1995:391723 BIOSIS
DN PREV199598406023
TI Common virulence factors for bacterial ***pathogenicity*** in plants and animals.
AU Rahme, Laurence G.; Stevens, Emily J.; Wolfort, Sean F.; Shao, Jing; Tompkins, Ronald G.; ***Ausubel, Frederick M. (1)***
CS (1) Dep. Genetics, Harvard Med. Sch., Boston, MA 02114 USA
SO Science (Washington D.C), (1995) Vol. 268, No. 5219, pp. 1899-1902.
ISSN: 0036-8075.
DT Article
LA English
AB A Pseudomonas aeruginosa strain (UCBPP-PA14) is infectious both in an Arabidopsis thaliana leaf infiltration model and in a mouse full-thickness skin burn model. UCBPP-PA14 exhibits ecotype specificity for Arabidopsis, causing a range of symptoms from none to severe in four different ecotypes. In the mouse model, UCBPP-PA14 is as lethal as other well-studied P. aeruginosa strains. Mutations in the UCBPP-PA14 toxA, plcS, and gacA genes resulted in a significant reduction in ***pathogenicity*** in both hosts, indicating that these genes encode virulence factors required for

the full expression of ***pathogenicity*** in both plants and animals.

L3 ANSWER 14 OF 34 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1995:304688 BIOSIS
DN PREV199598318988
TI Molecular genetics of plant disease resistance.
AU Staskawicz, Brian J. (1); ***Ausubel, Frederick M.*** ; Baker, Barbara J.; Ellis, Jeffrey G.; Jones, Jonathan D. G.
CS (1) Dep. Plant Biol., Univ. Calif., Berkeley, CA 94720 USA
SO Science (Washington D C), (1995) Vol. 268, No. 5211, pp. 661-667. ISSN: 0036-8075.
DT General Review
LA English
AB Plant breeders have used disease resistance genes (R genes) to control plant disease since the turn of the century. Molecular cloning of R genes that enable plants to resist a diverse range of ***pathogens*** has revealed that the proteins encoded by these genes have several features in common. These findings suggest that plants may have evolved common signal transduction mechanisms for the expression of resistance to a wide range of unrelated ***pathogens*** . Characterization of the molecular signals involved in ***pathogen*** recognition and of the molecular events that specify the expression of resistance may lead to novel strategies for plant disease control.

L3 ANSWER 15 OF 34 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1995:272740 BIOSIS
DN PREV199598287040
TI Use of Arabidopsis thaliana defense-related mutants to dissect the plant response to ***pathogens*** .
AU ***Ausubel, Frederick M. (1)*** ; Katagiri, Fumiaki; Mindrinos, Michael; Glazebrook, Jane
CS (1) Dep. Genetics, Harvard Med. Sch., Massachusetts General Hosp., Boston, MA 02114 USA
SO Proceedings of the National Academy of Sciences of the United States of America, (1995) Vol. 92, No. 10, pp. 4189-4196. ISSN: 0027-8424.
DT Article
LA English
AB The plant defense response to microbial ***pathogens*** had been studied primarily by using biochemical and physiological techniques. Recently, several laboratories have developed a variety of pathosystems utilizing Arabidopsis thaliana as a model host so that genetic analysis could also be used to study plant defense

responses. Utilizing a pathosystem that involves the infection of Arabidopsis with ***pathogenic*** pseudomonads, we have cloned the Arabidopsis disease-resistance gene RPS2, which corresponds to the avirulence gene avrRpt2 in a gene-for-gene relationship. RPS2 encodes a 105-kDa protein containing a leucine zipper, a nucleotide binding site, and 14 imperfect leucine-rich repeats. The RPS2 protein is remarkably similar to the product of the tobacco N gene, which confers resistance to tobacco mosaic virus. We have also isolated a series of Arabidopsis mutants that synthesize decreased levels of an Arabidopsis phytoalexin called camalexin. Analysis of these mutants indicated that camalexin does not play a significant role in limiting growth of avirulent Pseudomonas syringae strains during the hypersensitive defense response but that it may play a role in limiting the growth of virulent strains. More generally, we have shown that we can utilize Arabidopsis to systematically dissect the defense response by isolation and characterization of appropriate defense-related mutants.

L3 ANSWER 16 OF 34 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1995:136346 BIOSIS
DN PREV199598150646
TI Microbial ***Pathogenesis*** of Arabidopsis.
AU Crute, Ian (1); Beynon, Jim; Dangl, Jeff; Holub, Eric; Mauch-Mani, Brigitte; Slusarenko, Alan; Staskawicz, Brian; ***Ausubel, Fred***
CS (1) Plant Pathol. Weed Sci. Dep., Horticulture Res. Int., Wellesbourne, Warwick CV35 9EF UK
SO Meyerowitz, E. M. [Editor]; Somerville, C. R. [Editor]. Cold Spring Harbor Monograph Series, (1994) No. 27, pp. 705-747. Cold Spring Harbor Monograph Series; Arabidopsis.
Publisher: Cold Spring Harbor Laboratory Press 10 Skyline Drive, Plainview, New York 11803, USA.
ISSN: 0270-1847. ISBN: 0-87969-428-9.
DT Book
LA English

L3 ANSWER 17 OF 34 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1994:532260 BIOSIS
DN PREV199497545260
TI The A. thaliana disease resistance gene RPS2 encodes a protein containing a nucleotide-binding site and leucine-rich repeats.
AU Mindrinos, Michael (1); Katagiri, Fumiaki (1); Yu, Guo-Liang; ***Ausubel, Frederick M.***
CS (1) Dep. Genet., Harvard Med. Sch., Mass. Gen. Hosp., Boston, MA 02114 USA
SO Cell, (1994) Vol. 78, No. 6, pp. 1089-1099.

ISSN: 0092-8674.
DT Article
LA English
AB In plants, resistance to a ***pathogen*** is frequently correlated with a genetically defined interaction between a plant resistance gene and a corresponding ***pathogen*** avirulence gene. A simple model explains these gene-for-gene interactions: avirulence gene products generate signals (ligands), and resistance genes encode cognate receptors. The A. thaliana RPS2 gene confers resistance to the bacterial ***pathogen*** P. syringes carrying the avirulence gene avrRpt2. A map-based positional cloning strategy was used to identify RPS2. The identification of RPS2 was verified using a newly developed transient assay for RPS2 function and by genetic complementation In transgenic plants. RPS2 encodes a novel 105 kDa protein containing a leucine zipper, a nucleotide-binding site, and 14 imperfect leucine-rich repeats.

L3 ANSWER 18 OF 34 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1994:489601 BIOSIS
DN PREV199497502601
TI Isolation of phytoalexin-deficient mutants of Arabidopsis thaliana and characterization of their interactions with bacterial ***pathogens*** .
AU Glazebrook, Jane (1); ***Ausubel, Frederick M.***
CS (1) Dep. Genetics, Harvard Med. Sch., Massachusetts Gen. Hosp., Boston, MA 02114 USA
SO Proceedings of the National Academy of Sciences of the United States of America, (1994) Vol. 91, No. 19, pp. 8955-8959.
ISSN: 0027-8424.
DT Article
LA English
AB A genetic approach was used to assess the extent to which a particular plant defense response, phytoalexin biosynthesis, contributes to Arabidopsis thaliana resistance to Pseudomonas syringae ***pathogens*** . The A. thaliana phytoalexin, camalexin, accumulated in response to infection by various P. syringae strains. No correlation between ***pathogen*** avirulence and camalexin accumulation was observed. A biochemical screen was used to isolate three mutants of A. thaliana ecotype Columbia that were phytoalexin deficient (pad mutants). The mutations pad1, pad2, and pad3 were found to be recessive alleles of three different genes. pad1 and pad2 were mapped to chromosome IV and pad3 was mapped to chromosome III. Infection of pad mutant plants with strains carrying cloned avirulence genes revealed that the pad mutations did not affect the plants' ability to restrict the

growth of these strains. This result strongly suggests that in A. thaliana, phytoalexin biosynthesis is not required for resistance to avirulent P. syringae ***pathogens*** . Two of the pad mutants displayed enhanced sensitivity to isogenic virulent P. syringae ***pathogens*** , suggesting that camalexin may serve to limit the growth of virulent bacteria.

L3 ANSWER 19 OF 34 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1994:320325 BIOSIS
DN PREV199497333325
TI Programmed cell death in plants: A ***pathogen*** -triggered response activated coordinately with multiple defense functions.
AU Greenberg, Jean T. (1); Guo, Ailan; Klessig, Daniel F.; ***Ausubel, Frederick M. (1)***
CS (1) Dep. Genet., Harvard Med. Sch., Dep. Mol. Biol., Massachusetts Gen. Hosp., Boston, MA 02114 USA
SO Cell, (1994) Vol. 77, No. 4, pp. 551-563.
ISSN: 0092-8674.
DT Article
LA English
AB In plants, the hypersensitive response (HR) to ***pathogens*** involves rapid cell death, which is hypothesized to arise from the activation of a cell death program. We describe mutant A. thaliana plants that contain lesions in a single accelerated cell death (ACD) gene called ACD2 and that bypass the need for ***pathogen*** exposure to induce the HR, acd2 plants that develop spontaneous lesions show typical HR characteristics both within the necrotic tissue and within the healthy part of the plant, including: modification of plant cell walls, resistance to bacterial ***pathogens*** , and accumulation of defense-related gene transcripts, the signal molecule salicylic acid and an antimicrobial compound. We propose that the ACD2 gene is involved in a pathway(s) that negatively regulates a genetically programmed HR.

L3 ANSWER 20 OF 34 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1993:424760 BIOSIS
DN PREV199345072385
TI Analysis of the Arabidopsis defense response to Pseudomonas ***pathogens*** .
AU ***Ausubel, Frederick M. (1)*** ; Glazebrook, Jane; Greenberg, Jean; Mindrinos, Michael; Yu, Guo-Liang
CS (1) Dep. Genet., Harvard Med. Sch., Boston, MA 02114 USA
SO Nester, E. W. [Editor]; Verma, D. P. S. [Editor]. Current Plant Science and Biotechnology in Agriculture, (1993) Vol. 14, pp. 393-403. Current Plant Science and Biotechnology in Agriculture;

Advances in molecular genetics of plant-microbe interactions, Vol. 2.
Publisher: Kluwer Academic Publishers PO Box 989, 3300 AZ Dordrecht, Netherlands.
Meeting Info.: 6th International Symposium on Molecular Plant-Microbe Interactions Seattle, Washington, USA July 1992
ISSN: 0924-1949. ISBN: 0-7923-2045-X.
DT Article
LA English

L3 ANSWER 21 OF 34 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1993:241799 BIOSIS
DN PREV199344114999
TI Analysis of the Arabidopsis defense response to Pseudomonas ***pathogens*** .
AU ***Ausubel, Frederick M.*** ; Glazebrook, Jane; Greenberg, Jean; Katagiri, Fumiaki; Mindrinos, Michael; Yu, Guo-Liang
CS Dep. Genet., Harvard Med. Sch., Boston, MA 02114 USA
SO Journal of Cellular Biochemistry Supplement, (1993) Vol. 0, No. 17 PART C, pp. 129.
Meeting Info.: Keystone Symposium on Molecular Genetic Controls of Microbial Differentiation Tamarron, Colorado, USA February 17-23, 1993
ISSN: 0733-1959.
DT Conference
LA English

L3 ANSWER 22 OF 34 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1993:207375 BIOSIS
DN PREV199395108600
TI An Arabidopsis thaliana lipoxygenase gene can be induced by ***pathogens*** , abscisic acid, and methyl jasmonate.
AU Melan, Melissa A.; Dong, Xinnian; Endara, Mirei E.; Davis, Keith R.; ***Ausubel, Frederick M.*** ; Peterman, T. Kaye (1)
CS (1) Dep. Biol. Sciences, Wellesley College, Wellesley, MA 02181 USA
SO Plant Physiology (Rockville), (1993) Vol. 101, No. 2, pp. 441-450.
ISSN: 0032-0889.
DT Article
LA English
AB We isolated and characterized a 2.8-kb, full-length, Arabidopsis thaliana cDNA clone encoding a lipoxygenase. DNA sequence analysis showed that deduced amino acid sequence of the Arabidopsis protein is 72 to 78% similar to that of legume seed lipoxygenases. DNA blot analysis indicated that Arabidopsis contains a single gene, LOX1, with appreciable homology to the cDNA clone. RNA blot analysis

showed that the LOX1 gene is expressed in Arabidopsis leaves, roots, inflorescences, and young seedlings. LOX1 expression levels were highest in roots and young seedlings. In mature plants, LOX1 mRNA levels increased upon treatment with the stress-related hormones abscisic acid and methyl jasmonate and remained high for at least 96 h. Expression of the LOX1 gene was examined following infiltration of leaves with virulent (Psm ES4326) and avirulent (Pst MM1065) strains of Pseudomonas syringae. LOX1 mRNA levels were induced approximately 6-fold by both virulent and avirulent strains; however, the response to avirulent strains was much more rapid. Infiltration of leaves with Pst MM1065 resulted in maximal induction within 12 h, whereas maximal induction by Psm ES4326 did not occur until 48 h. When a cloned avr gene, avrRpt2, was transferred to Psm ES4326, LOX1 mRNA accumulated in a pattern similar to that observed for the avirulent strain Pst MM1065.

L3 ANSWER 23 OF 34 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1993:75614 BIOSIS
DN PREV199395040114
TI Large, identical, tandem repeating units in the C protein alpha antigen gene, bca, of group B streptococci.
AU Michel, James L. (1); Madoff, Lawrence C.; Olson, Kristin; Kling, David E.; Kasper, Dennis L.; ***Ausubel, Frederick M.***
CS (1) Channing Lab., 180 Longwood Ave., Boston, Mass. 02115 USA
SO Proceedings of the National Academy of Sciences of the United States of America, (1992) Vol. 89, No. 21, pp. 10060-10064.
ISSN: 0027-8424.
DT Article
LA English
AB Group B Streptococcus (GBS) (Streptococcus agalactiae) is the leading cause of neonatal sepsis and meningitis in the United States. The surface-associated C protein alpha antigen of GBS is thought to have a role in both virulence and immunity. We previously cloned the C protein alpha antigen structural gene (named bca for group B, C protein, alpha) into Escherichia coli. Western blots of both the native alpha antigen and the cloned gene product demonstrate a regularly laddered pattern of heterogeneous polypeptides. The nucleotide sequence of the bca locus reveals an open reading frame of 3060 nucleotides encoding a precursor protein of 108,705 Da. Cleavage of a putative signal sequence of 41 amino acids yields a mature protein of 104,106 Da. The 20,417-Da N-terminal region of the alpha antigen shows no homology to previously described protein sequences and is followed by a series of nine tandem repeating units that make up 74% of the mature protein. Each repeating unit is identical and consists of 82 amino

acids with a molecular mass of 8665 Da, which is encoded by 246 nucleotides. The size of the repeating units corresponds to the observed size differences in the heterogeneous ladder of alpha C proteins expressed by GBS. The C-terminal region of the alpha antigen contains a membrane anchor domain motif that is shared by a number of Gram-positive surface proteins. The large region of identical repeating units in bca defines protective epitopes and may play a role in generating phenotypic and genotypic diversity of the alpha antigen.

L3 ANSWER 24 OF 34 CAPLUS COPYRIGHT 1998 ACS
AN 1998:784293 CAPLUS
TI Modulation of expression of the ToxR regulon in Vibrio cholerae by a member of the two-component family of response regulators
AU Wong, Sandy M.; Carroll, Patricia A.; Rahme, Laurence G.; ***Ausubel, Frederick M.*** ; Calderwood, Stephen B.
CS Department of Molecular Biology, Infectious Disease Division, Massachusetts General Hospital, Boston, MA, 02114, USA
SO Infect. Immun. (1998), 66(12), 5854-5861
CODEN: INFIBR; ISSN: 0019-9567
PB American Society for Microbiology
DT Journal
LA English
AB The ToxRS system in Vibrio cholerae plays a central role in the modulation of virulence gene expression in response to environmental stimuli. An integration of multiple signalling inputs mediated by ToxR, -S, and -T controls virulence gene expression leading to cholera toxin (CT) prodn. Recently, we identified a new virulence locus, varA (virulence assocd. regulator), in classical V. cholerae O1 that pos. controls transcription of tcpA, the major subunit of the toxin-coregulated pilus (TCP) and the prodn. of CT, two key factors in cholera ***pathogenesis*** . The varA locus is a homolog of gacA (originally described for the soil organism Pseudomonas fluorescens), which encodes a conserved global regulator belonging to the family of two-component signal transducing mols. GacA homologs in a no. of diverse gram-neg. ***pathogenic*** bacterial species have been implicated in controlling the prodn. of diverse virulence factors. VarA mutants showed reduced levels of tcpA message and TcpA protein, lacked visible signs of autoagglutination (a phenotype assocd. with functional TCP), produced decreased levels of CT, and were attenuated in colonizing infant mice. Transcription of varA appears to be independent of ToxR, and overexpression of the regulators tcpPH and toxT from plasmids in the varA mutant restored wild-type levels of CT prodn. and the ability to autoagglutinate. VarA represents an addnl.

modulating factor in the coordinate expression of virulence factors in V. cholerae.

L3 ANSWER 25 OF 34 CAPLUS COPYRIGHT 1998 ACS
AN 1998:744978 CAPLUS
DN 130:2014
TI Methods of screening compounds useful for prevention of infection or ***pathogenicity***
IN ***Ausubel, Frederick M.*** ; Rahme, Lawrence G.; Tan, Man-wah; Ruvkun, Gary B.; Mahajan-Miklos, Shalina; Broeks, Annegien; Plasterk, Ronald H. A.; Jander, Georg; Heard, Jacqueline
PA The General Hospital Corp., USA; The Netherlands Cancer Institute
SO PCT Int. Appl., 87 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9850080	A1	19981112	WO 98-US9150	19980508

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

PRAI US 97-852927 19970508
US 97-962750 19971103
AB Screening procedures are disclosed for identifying compds. useful for inhibiting infection or ***pathogenicity*** . Methods are also disclosed for identifying ***pathogenic*** virulence factors.

L3 ANSWER 26 OF 34 CAPLUS COPYRIGHT 1998 ACS
AN 1998:126277 CAPLUS
DN 128:201804
TI Acquired resistance NPR1 genes from Arabidopsis thaliana and Nicotiana glutinosa and their use for genetic engineering
IN ***Ausubel, Frederick M.*** ; Glazebrook, Jane; Dong, Xinnian; Cao, Hui
PA General Hospital Corporation, USA; Duke University
SO PCT Int. Appl., 128 pp.

CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9806748	A1	19980219	WO 97-US13994	19970808
AU 9739128	A1	19980306	AU 97-39128	19970808

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

PRAI US 96-23851 19960809
US 97-35166 19970110
US 97-46769 19970516
WO 97-US13994 19970808

AB Genomic and cDNA sequences encoding plant acquired resistance proteins are provided from cruciferous (Arabidopsis thaliana) and solanaceous (Nicotiana glutinosa) plants. Npr mutants showed that the NPR1 gene of A. thaliana is active in controlling the defense response against a broad spectrum of ***pathogens*** , and the gene was cloned using a map-based positional cloning strategy. The NPR1 protein comprised 593 amino acid residues and contained ankyrin-repeat and G-protein coupled receptor motifs as well as nuclear localization signals. NPR1 mediates the expression of ***pathogenesis*** -related polypeptides. Expression of these polypeptides in transgenic plants are useful for providing enhanced defense mechanisms to combat plant diseases.

L3 ANSWER 27 OF 34 CAPLUS COPYRIGHT 1998 ACS
AN 1996:679267 CAPLUS
DN 125:317307
TI Bioassay methods using different eukaryotic organisms for screening compounds useful for prevention of infection or ***pathogenicity*** and for identifying ***pathogenic*** virulence factors
IN ***Ausubel, Frederick M.*** ; Rahme, Laurence G.; Tan, Man-Wah; Ruvkun, Gary B.
PA General Hospital Corporation, USA
SO PCT Int. Appl., 50 pp.

CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9630053	A1	19961003	WO 96-US4210	19960327
W: CA, JP				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
CA 2215836	AA	19961003	CA 96-2215836	19960327
EP 828520	A1	19980318	EP 96-911451	19960327
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				

PRAI US 95-411560 19950328
WO 96-US4210 19960327
AB Screening procedures are disclosed for identifying compds. useful for inhibiting infection or ***pathogenicity*** . The methodol. of the invention involves (a) exposing .gtoreq.2 different eukaryotic organisms, .gtoreq.1 of which is a non-rodent, to a single ***pathogen*** in the presence of .gtoreq.1 candidate compd; and (b) identifying a compd. that inhibits the ***pathogen*** in each of the eukaryotic organisms. Methods are also disclosed for identifying ***pathogenic*** virulence factors.

L3 ANSWER 28 OF 34 CAPLUS COPYRIGHT 1998 ACS
AN 1995:997362 CAPLUS
DN 124:47615
TI RPS gene family, primers, probes, detection methods, and use in transgenic plant disease resistance to ***pathogen***
IN ***Ausubel, Frederick M.*** ; Staskawicz, Brian J.; Bent, Andrew F.; Dahlbeck, Douglas; Katagiri, Fumiaki; Kunkel, Barbara N.; Mindrinos, Michael N.; Yu, Guo-liang; Baker, Barbara; et al.
PA General Hospital Corp., USA; University of California; United States Department of Agriculture; Commonwealth Scientific and Industrial Research Organization
SO PCT Int. Appl., 187 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9528423	A1	19951026	WO 95-US4589	19950413

W: AM, AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LT, LU, LV, MD, MG, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TT, UA
RW: KE, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

CA 2187771 AA 19951026 CA 95-2187771 19950413
AU 9523565 A1 19951110 AU 95-23565 19950413
AU 693891 B2 19980709
EP 763058 A1 19970319 EP 95-917564 19950413
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, NL, PT, SE

PRAI US 94-227360 19940413
US 94-310912 19940922
WO 95-US4589 19950413

OS MARPAT 124:47615

AB Disclosed is substantially pure DNA encoding an Arabidopsis thaliana Rps2 polypeptide; substantially pure Rps2 polypeptide; and methods of using such DNA to express the Rps2 polypeptide in plant cells and whole plants to provide, in transgenic plants, disease resistance to ***pathogens*** . Also disclosed are conserved regions characteristic of the RPS family and primers and probes for the identification and isolation of addnl. RPS disease-resistance genes.

L3 ANSWER 29 OF 34 CAPLUS COPYRIGHT 1998 ACS
AN 1995:997356 CAPLUS
DN 124:51008
TI Rps2 gene of Arabidopsis thaliana and its use for preparing diseases-resistant transgenic plants
IN ***Ausubel, Frederick M.*** ; Staskawicz, Brian J.; Bent, Andrew F.; Dahlbeck, Douglas; Katagiri, Fumiaki; Kunkel, Barbara N.; Mindrinos, Michael N.; Yu, Guo-liang
PA General Hospital Corp., USA; University of California
SO PCT Int. Appl., 88 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9528478	A1	19951026	WO 95-US4570	19950413

W: AM, AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LT, LU, LV, MD, MG, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG,

SI, SK, TJ, TT, UA
RW: KE, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG
CA 2187546 AA 19951026 CA 95-2187546 19950413
AU 9522895 A1 19951110 AU 95-22895 19950413
EP 759068 A1 19970226 EP 95-916372 19950413
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, NL, PT, SE
CN 1151183 A 19970604 CN 95-193569 19950413
HU 76257 A2 19970728 HU 96-2809 19950413
JP 09511909 T2 19971202 JP 95-527087 19950413
PRAI US 94-227360 19940413
WO 95-US4570 19950413
AB Disclosed is substantially pure DNA encoding an Arabidopsis thaliana rps2 polypeptide; substantially pure rps2 polypeptide; and methods of using such DNA to express the rps2 polypeptide in plant cells and whole plants to provide, in transgenic plants, disease resistance to ***pathogens*** .

L3 ANSWER 30 OF 34 CAPLUS COPYRIGHT 1998 ACS
AN 1995:462613 CAPLUS
DN 122:235173
TI Microbial ***pathogenesis*** of Arabidopsis
AU Crute, Ian; Beynon, Jim; Dangl, Jeff; Holub, Eric; Mauch-Mani, Brigitte; Slusarenko, Alan; Staskawicz, Brian; ***Ausubel, Fred***
CS Plant Pathology and Weed Science Department, Horticulture Research International, Wellesbourne/Warwick, CV35 9EF, UK
SO Cold Spring Harbor Monogr. Ser. (1994), 27, 705-47
CODEN: CHMSDK; ISSN: 0270-1847
DT Journal; General Review
LA English
AB A review with >100 refs.

L3 ANSWER 31 OF 34 CAPLUS COPYRIGHT 1998 ACS
AN 1991:675979 CAPLUS
DN 115:275979
TI Differential induction of 3-deoxy-D-arabino-heptulosonate 7-phosphate synthase genes in Arabidopsis thaliana by wounding and ***pathogenic*** attack
AU Keith, Brian; Dong, Xinnian; ***Ausubel, Frederick M.*** ; Fink, Gerald R.
CS Nine Cambridge Cent., Whitehead Inst. Biomed. Res., Cambridge, MA, 02142, USA
SO Proc. Natl. Acad. Sci. U. S. A. (1991), 88(19), 8821-5

CODEN: PNASA6; ISSN: 0027-8424
DT Journal
LA English
AB CDNAs from 2 distinct genes encoding 3-deoxy-D-arabino-heptulosonate 7-phosphate (DAHP) synthase (EC 4.1.2.15) in A. thaliana were isolated. Predicted protein sequences from both genes, DHS1 and DHS2, and a potato DAHP synthase gene are highly related, but none shows significant similarity to conserved microbial DAHP synthase proteins. Despite this structural difference, the DSH1 cDNA complements mutations in a yeast strain lacking DAHP synthase activity. DSH1 RNA levels increase in Arabidopsis leaves subjected either to phys. wounding or to infiltration with ***pathogenic*** Pseudomonas syringae strains. DHS2 RNA levels are not increased by these treatments, suggesting that the DHS1 and DHS2 proteins fulfill different physiol. functions. Other enzymes in the Arabidopsis arom. pathway are also encoded by duplicated genes, an arrangement that may allow independent regulation of arom. amino acid biosynthesis by distinct physiol. requirements such as protein synthesis and secondary metab. The presence of amino-terminal extensions characteristic of chloroplast transit peptides on DHS1 and DHS2 suggests that both proteins may be targeted to the chloroplast.

L3 ANSWER 32 OF 34 CAPLUS COPYRIGHT 1998 ACS
AN 1991:675977 CAPLUS
DN 115:275977
TI Virulence of selected phytopathogenic pseudomonads in Arabidopsis thaliana
AU Davis, Keith R.; Schott, Eric; ***Ausubel, Frederick M.***
CS Ohio State Biotechnol. Cent., Columbus, OH, 43210, USA
SO Mol. Plant-Microbe Interact. (1991), 4(5), 477-88
CODEN: MPMIEL; ISSN: 0894-0282
DT Journal
LA English
AB A model ***pathogenesis*** system is developed that utilizes A. thaliana as a host for infection by a variety of phytopathogenic Xanthomonas and Pseudomonas strains. Fifty-one different strains were divided into 4 categories based on the symptoms they elicited when infiltrated into A. thaliana leaves at 2 doses, 106 and 107 cfu/mL. Highly virulent and weakly virulent strains elicited spreading water-soaked lesions within 48 h at doses of 106 and 107 cfu/mL, resp. Avirulent strains elicited either a hypersensitive response (dry necrotic lesion) within 12-24 h at doses of 106 and 107 cfu/mL or pitting with mild chlorosis within 48 h at a dose of 107 cfu/mL. Null strains elicited no visible symptoms at a dose of

107 cfu/mL. Several Pseudomonas strains were chosen for addnl. characterization. The highly virulent strains, P. syringae pv. tomato DC3000 and P. syringae pv. maculicola 795, elicited spreading water-soaked lesions with chlorotic margins, multiplied 103- to 104-fold in A. thaliana leaves, induced a 5-10-fold transient accumulation of mRNA corresponding to phenylalanine ammonia-lyase (PAL), and had little effect on the pH of the medium when added to Arabidopsis tissue culture cells. The avirulent strain, P. cichorii 83-1, elicited a localized, dry lesion typical of hypersensitive response within 12-24 h, failed to multiply in A. thaliana leaves, induced a 15-30-fold accumulation of PAL mRNA, and caused an alkalinization of the medium when added to Arabidopsis tissue cultures. P. aureofaciens strain 923 elicited a null response, did not multiply in A. thaliana leaves, induced a 5-6-fold accumulation of PAL mRNA, and caused an acidification of the medium when added to Arabidopsis tissue culture cells. To facilitate the monitoring of the induction of A. thaliana PAL mRNA in response to infiltration with phytopathogenic bacteria, an A. thaliana genomic DNA sequence encoding PAL was cloned and partially sequenced. Southern blot anal. showed that the A. thaliana genome contains 2 or 3 addnl. sequences that are at least partially homologous to the cloned PAL gene.

L3 ANSWER 33 OF 34 CAPLUS COPYRIGHT 1998 ACS
AN 1991:402467 CAPLUS
DN 115:2467
TI Induction of Arabidopsis defense genes by virulent and avirulent Pseudomonas syringae strains and by a cloned avirulence gene
AU Dong, Xinnian; Mindrinos, Michael; Davis, Keith R.; ***Ausubel,*** *** Frederick M.***
CS Dep. Genet., Harvard Med. Sch., Boston, MA, 02114, USA
SO Plant Cell (1991), 3(1), 61-72
CODEN: PLCEEW; ISSN: 1040-4651
DT Journal
LA English
AB A model system was developed to study the signal transduction pathways leading to the activation of A. thaliana genes involved in the defense against ***pathogen*** attack. Here, the identification and characterization of virulent and avirulent P. syringae strains that elicit disease or resistance symptoms when infiltrated into Arabidopsis leaves are described. The virulent and avirulent strains were characterized by detg. growth of the ***pathogen*** in Arabidopsis leaves and by measuring accumulation of mRNA corresponding to Arabidopsis phenylalanine ammonia-lyase (PAL), .beta.-1,3-glucanase (BG), and chalcone synthase (CHS) genes

in infected leaves. The virulent strain, P. syringae pv maculicola ES4326, multiplied 105-fold in Arabidopsis leaves and strongly elicited BG1, BG2, and BG3 mRNA accumulation but had only a modest effect on PAL mRNA accumulation. In contrast, the avirulent strain, P. syringae pv tomato MM1065, multiplied less than 10-fold in leaves and had only a minimal effect on BG1, BG2, and BG3 mRNA accumulation, but it induced PAL mRNA accumulation. No accumulation of CHS mRNA was found with either ES4326 or MM1065. The cloning of a putative avirulence (avr) gene from the avirulent strain MM1065 that caused the virulent strain ES4326 to grow less well in leaves and to strongly elicit PAL but not BG1 and BG3 mRNA accumulation is also discussed. These results suggest that the Arabidopsis PAL and BG genes may be activated by distinct signal transduction pathways and show that differences in plant gene induction by virulent and avirulent strains can be attributed to a cloned presumptive avr gene.

L3 ANSWER 34 OF 34 CAPLUS COPYRIGHT 1998 ACS
AN 1989:491690 CAPLUS
DN 111:91690
TI General method for site-directed mutagenesis in Escherichia coli O18ac:K1:H7: deletion of the inducible superoxide dismutase gene, sodA, does not diminish bacteremia in neonatal rats
AU Bloch, Craig A.; Thorne, Grace M.; ***Ausubel, Frederick M.***
CS Dep. Genet., Harvard Med. Sch., Boston, MA, 02114, USA
SO Infect. Immun. (1989), 57(7), 2141-8
CODEN: INFIBR; ISSN: 0019-9567
DT Journal
LA English
AB A defined deletion in the E. coli K-12 sodA gene (encoding manganese-superoxide dismutase) linked to a nontransposable selectable marker was generated by transposon Tn5 insertion in combination with in vitro mutagenesis. This mutant allele was used to replace the wild-type sodA gene in an E. coli clin. isolate of serotype O18ac:K1:H7 by bacteriophage P1 transduction. The O18ac:K1:H7 sodA mutant contained no manganese-superoxide dismutase and no hybrid manganese-iron-superoxide dismutase. The sodA mutant was more sensitive to paraquat toxicity than were the parental strain and an isogenic mutant bearing an analogously constructed sodA+ Tn5 insertion allele. In a suckling rat model of bacteremia following oral inoculation of E. coli K1, the sodA mutant was undiminished in its capabilities both to colonize the gastrointestinal tract and, surprisingly, to cause bacteremia. In conjunction with the rat model for E. coli K1 ***pathogenesis*** , the method for site-directed mutagenesis described in this paper

permits detn. of the role played in colonization and bacteremia by any K1 gene which either has a homolog in E. coli K-12 or can be cloned and manipulated therein.

=> e rahme laurence/au

E1 2 RAHME L/AU
E2 30 RAHME L G/AU
E3 0 --> RAHME LAURENCE/AU
E4 9 RAHME LAURENCE G/AU
E5 1 RAHME LAWRENCE G/AU
E6 13 RAHME M/AU
E7 2 RAHME M M/AU
E8 1 RAHME MARC/AU
E9 1 RAHME MAROUN/AU
E10 1 RAHME MAROUN GEORGE/AU
E11 4 RAHME N/AU
E12 1 RAHME R W/AU

=> s e1-e5

L4 42 ("RAHME L"/AU OR "RAHME L G"/AU OR "RAHME LAURENCE"/AU OR "RAHME LAURENCE G"/AU OR "RAHME LAWRENCE G"/AU)

=> dup rem l4

PROCESSING COMPLETED FOR L4
L5 14 DUP REM L4 (28 DUPLICATES REMOVED)

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YOU HAVE REQUESTED DATA FROM 14 ANSWERS - CONTINUE? Y/(N):y

L5 ANSWER 1 OF 14 CAPLUS COPYRIGHT 1998 ACS
AN 1998:744978 CAPLUS
DN 130:2014
TI Methods of screening compounds useful for prevention of infection or pathogenicity
IN Ausubel, Frederick M.; ***Rahme, Lawrence G.*** ; Tan, Man-wah; Ruvkun, Gary B.; Mahajan-Miklos, Shalina; Broeks, Annegien; Plasterk, Ronald H. A.; Jander, Georg; Heard, Jacqueline
PA The General Hospital Corp., USA; The Netherlands Cancer Institute
SO PCT Int. Appl., 87 pp.
CODEN: PIXXD2

DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9850080	A1	19981112	WO 98-US9150	19980508

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

PRAI US 97-852927 19970508
US 97-962750 19971103

AB Screening procedures are disclosed for identifying compds. useful for inhibiting infection or pathogenicity. Methods are also disclosed for identifying pathogenic virulence factors.

L5 ANSWER 2 OF 14 CAPLUS COPYRIGHT 1998 ACS DUPLICATE 1
AN 1998:784293 CAPLUS
TI Modulation of expression of the ToxR regulon in Vibrio cholerae by a member of the two-component family of response regulators
AU Wong, Sandy M.; Carroll, Patricia A.; ***Rahme, Laurence G.*** ; Ausubel, Frederick M.; Calderwood, Stephen B.
CS Department of Molecular Biology, Infectious Disease Division, Massachusetts General Hospital, Boston, MA, 02114, USA
SO Infect. Immun. (1998), 66(12), 5854-5861
CODEN: INFIBR; ISSN: 0019-9567
PB American Society for Microbiology
DT Journal
LA English
AB The ToxRS system in Vibrio cholerae plays a central role in the modulation of virulence gene expression in response to environmental stimuli. An integration of multiple signalling inputs mediated by ToxR, -S, and -T controls virulence gene expression leading to cholera toxin (CT) prodn. Recently, we identified a new virulence locus, varA (virulence assocd. regulator), in classical V. cholerae O1 that pos. controls transcription of tcpA, the major subunit of the toxin-coregulated pilus (TCP) and the prodn. of CT, two key factors in cholera pathogenesis. The varA locus is a homolog of gacA (originally described for the soil organism Pseudomonas fluorescens), which encodes a conserved global regulator belonging

to the family of two-component signal transducing mols. GacA homologs in a no. of diverse gram-neg. pathogenic bacterial species have been implicated in controlling the prodn. of diverse virulence factors. VarA mutants showed reduced levels of tcpA message and TcpA protein, lacked visible signs of autoagglutination (a phenotype assocd. with functional TCP), produced decreased levels of CT, and were attenuated in colonizing infant mice. Transcription of varA appears to be independent of ToxR, and overexpression of the regulators tcpPH and toxT from plasmids in the varA mutant restored wild-type levels of CT prodn. and the ability to autoagglutinate. VarA represents an addnl. modulating factor in the coordinate expression of virulence factors in V. cholerae.

L5 ANSWER 3 OF 14 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 2
AN 1998:49050 BIOSIS
DN PREV199800049050
TI Use of model plant hosts to identify Pseudomonas aeruginosa virulence factors.
AU ***Rahme, Laurence G.*** ; Tan, Man-Wah; Le, Long; Wong, Sandy M.; Tompkins, Ronald G.; Calderwood, Stephen B.; Ausubel, Frederick M. (1)
CS (1) Dep. Genetics Harvard Med. Sch., Dep. Molecular Biol., Massachusetts General Hosp., Boston, MA 02114 USA
SO Proceedings of the National Academy of Sciences of the United States of America, (Nov. 25, 1997) Vol. 94, No. 24, pp. 13245-13250. ISSN: 0027-8424.
DT Article
LA English
AB We used plants as an in vivo pathogenesis model for the identification of virulence factors of the human opportunistic pathogen Pseudomonas aeruginosa. Nine of nine TnphoA mutant derivatives of P. aeruginosa strain UCBPPPA14 that were identified in a plant leaf assay for less pathogenic mutants also exhibited significantly reduced pathogenicity in a burned mouse pathogenicity model, suggesting that P. aeruginosa utilizes common strategies to infect both hosts. Seven of these nine mutants contain TnphoA insertions in previously unknown genes. These results demonstrate that an alternative nonvertebrate host of a human bacterial pathogen can be used in an in vivo high throughput screen to identify novel bacterial virulence factors involved in mammalian pathogenesis.

L5 ANSWER 4 OF 14 CAPLUS COPYRIGHT 1998 ACS
AN 1996:679267 CAPLUS
DN 125:317307
TI Bioassay methods using different eukaryotic organisms for screening

compounds useful for prevention of infection or pathogenicity and for identifying pathogenic virulence factors
IN Ausubel, Frederick M.; ***Rahme, Laurence G.*** ; Tan, Man-Wah; Ruvkun, Gary B.
PA General Hospital Corporation, USA
SO PCT Int. Appl., 50 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9630053	A1	19961003	WO 96-US4210	19960327
W: CA, JP				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
CA 2215836	AA	19961003	CA 96-2215836	19960327
EP 828520	A1	19980318	EP 96-911451	19960327
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				

PRAI US 95-411560 19950328
WO 96-US4210 19960327
AB Screening procedures are disclosed for identifying compds. useful for inhibiting infection or pathogenicity. The methodol. of the invention involves (a) exposing .gtoreq.2 different eukaryotic organisms, .gtoreq.1 of which is a non-rodent, to a single pathogen in the presence of .gtoreq.1 candidate compd; and (b) identifying a compd. that inhibits the pathogen in each of the eukaryotic organisms. Methods are also disclosed for identifying pathogenic virulence factors.

L5 ANSWER 5 OF 14 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 3
AN 1995:391723 BIOSIS
DN PREV199598406023
TI Common virulence factors for bacterial pathogenicity in plants and animals.
AU ***Rahme, Laurence G.*** ; Stevens, Emily J.; Wolfort, Sean F.; Shao, Jing; Tompkins, Ronald G.; Ausubel, Frederick M. (1)
CS (1) Dep. Genetics, Harvard Med. Sch., Boston, MA 02114 USA
SO Science (Washington D C), (1995) Vol. 268, No. 5219, pp. 1899-1902. ISSN: 0036-8075.
DT Article
LA English
AB A Pseudomonas aeruginosa strain (UCBPP-PA14) is infectious both in an Arabidopsis thaliana leaf infiltration model and in a mouse

full-thickness skin burn model. UCBPP-PA14 exhibits ecotype specificity for Arabidopsis, causing a range of symptoms from none to severe in four different ecotypes. In the mouse model, UCBPP-PA14 is as lethal as other well-studied P. aeruginosa strains. Mutations in the UCBPP-PA14 toxA, plcS, and gacA genes resulted in a significant reduction in pathogenicity in both hosts, indicating that these genes encode virulence factors required for the full expression of pathogenicity in both plants and animals.

L5 ANSWER 6 OF 14 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 4
AN 1993:424746 BIOSIS
DN PREV199345072371
TI Pseudomonas syringae pv. phaseolicola-plant interactions: Host-pathogen signalling through cascade control of hrp gene expression.
AU Miller, W. (1); Mindrinos, M. N.; ***Rahme, L. G.*** ; Frederick, R. D.; Grimm, C.; Gressman, R.; Kyriakides, X.; Kokkinidis, M.; Panopoulos, N. J. (1)
CS (1) Dep. Plant Pathol., Univ. Calif., Berkeley, CA USA
SO Nester, E. W. [Editor]; Verma, D. P. S. [Editor]. Current Plant Science and Biotechnology in Agriculture, (1993) Vol. 14, pp. 267-274. Current Plant Science and Biotechnology in Agriculture; Advances in molecular genetics of plant-microbe interactions, Vol. 2.
Publisher: Kluwer Academic Publishers PO Box 989, 3300 AZ Dordrecht, Netherlands.
Meeting Info.: 6th International Symposium on Molecular Plant-Microbe Interactions Seattle, Washington, USA July 1992
ISSN: 0924-1949. ISBN: 0-7923-2045-X.
DT Article
LA English

L5 ANSWER 7 OF 14 CAPLUS COPYRIGHT 1998 ACS
AN 1992:606050 CAPLUS
DN 117:206050
TI Genetic and transcriptional organization of the hrp cluster of Pseudomonas syringae pv. phaseolicola. [Erratum to document cited in CA114(11):95962z]
AU ***Rahme, Laurence G.*** ; Mindrinos, Michael N.; Panopoulos, Nickolas J.
CS Dep. Plant Pathol., Univ. California, Berkeley, CA, 94720, USA
SO J. Bacteriol. (1992), 174(11), 3840
CODEN: JOBAAY; ISSN: 0021-9193
DT Journal
LA English

AB Errors in Table 1 have been cor. The errors were not reflected in the abstr. or the index entries.

L5 ANSWER 8 OF 14 EMBASE COPYRIGHT 1998 ELSEVIER SCI. B.V.
AN 92214064 EMBASE
TI Erratum: Genetic and transcriptional organization of the hrp cluster of Pseudomonas syringae pv. phaseolicola (Journal of Bacteriology 173 (576)).
AU ***Rahme L.G.*** ; Mindrinos M.N.; Panopoulos N.J.
CS Department of Plant Pathology, University of California, Berkeley, CA 94720, United States
SO J. BACTERIOL., (1992) 174/11 (3840).
ISSN: 0021-9193 CODEN: JOBAAY
CY United States
DT Journal
FS 004 Microbiology
LA English

L5 ANSWER 9 OF 14 SCISEARCH COPYRIGHT 1998 ISI (R)
AN 92:350446 SCISEARCH
GA The Genuine Article (R) Number: HX278
TI CORRECTION
AU ***RAHME L G (Reprint)***
SO JOURNAL OF BACTERIOLOGY, (JUN 1992) Vol. 174, No. 11, pp. 3840.
ISSN: 0021-9193.
DT Errata; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 1

L5 ANSWER 10 OF 14 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 5
AN 1992:347152 BIOSIS
DN BA94:39377
TI PLANT AND ENVIRONMENTAL SENSORY SIGNALS CONTROL THE EXPRESSION OF
HRP GENES IN PSEUDOMONAS-SYRINGAE PV. PHASEOLICOLA.
AU ***RAHME L G*** ; MINDRINOS M N; PANOPOULOS N J
CS DEP. PLANT PATHOL., UNIV. CALIFORNIA, BERKELEY, CALIF. 94720.
SO J BACTERIOL, (1992) 174 (11), 3499-3507.
CODEN: JOBAAY. ISSN: 0021-9193.
FS BA; OLD
LA English
AB The hrp genes of Pseudomonas syringae pv. phaseolicola control the development of primary disease symptoms in bean plants and the elicitation of the hypersensitive response in resistant plants. We

examined the expression of the seven operons located in the 22-kb hrp cluster (L. G. Rahme, M. N. Mindrinos, and N. J. Panopoulos, J. Bacteriol. 173:575-586, 1991) in planta and in vitro under different physiological and nutritional conditions by using chromosomally located hrp::inaZ reporter fusions. We show that (i) a plant signal(s) is specifically required for the induction of the seven hrp operons, during both compatible and incompatible interactions; (ii) hrpL and hrpRS are regulated by different mechanisms in planta and in vitro; and (iii) expression of individual hrp loci is differentially affected by pH, osmotic strength, and type of carbon source: hrpAB, hrpC, and hrpD were downregulated similarly by osmolarity, pH, and certain carbon sources; hrpE expression was affected strongly by pH and carbon substrate and slightly by osmolarity; and hrpF was not substantially affected by any of these factors. These findings suggest complex signaling mechanisms taking place during plant-pathogen interactions.

L5 ANSWER 11 OF 14 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 6
AN 1991:135773 BIOSIS
DN BA91:72313
TI GENETIC AND TRANSCRIPTIONAL ORGANIZATION OF THE HRP CLUSTER OF PSEUDOMONAS-SYRINGAE PATHOVAR PHASEOLICOLA.
AU ***RAHME L G*** ; MINDRINOS M N; PANOPOULOS N J
CS DEP. PLANT PATHOLOGY, UNIVESITY CALIFORNIA, BERKELEY, CALIF. 94720.
SO J BACTERIOL, (1991) 173 (2), 575-586.
CODEN: JOBAAY. ISSN: 0021-9193.
FS BA; OLD
LA English
AB The hrp cluster of Pseudomonas syringae pv. phaseolicola encodes functions that are essential for pathogenicity on bean plants and for the elicitation of the hypersensitive response on resistant plants. The cluster was saturated with insertions of transposon Tn3-spice that served both as a mutagen and as a sensitive reporter of the expression of the target regions. The mutations covered a 17.5-kb segment in strain NPS3121, in which seven hrp::Tn5 insertions had been previously mapped, and regions outside this segment. The cluster is organized into seven distinct complementation groups (hrpL, hrpAB, hrpC, hrpE, hrpD, hrpE, hrpF, and hrpSR) on the basis of the analysis of over 100 Tn3-spice insertions in plasmids and 43 similar insertions in the chromosome; it spans nearly 22kb and is chromosomally located. The transcriptional orientation of all genes in the cluster was established by measuring the level of ice nucleation activity of complemented merodiploids carrying chromosomal hrp::inaZ fusion after inoculation in Red Kidney bean leaves. Although all seven loci

were actively expressed in Red Kidney bean leaves, none of them substantially expressed when the bacteria were grown in King B broth medium. Mutations in all loci, except those in hrpC, greatly reduced the ability of the bacteria to multiply in bean leaves. Mutations in the hrpC locus, although preventing the bacteria from eliciting a hypersensitive reaction on tobacco, allowed the bacteria to produce delayed and attenuated symptoms in Red Kidney bean leaves and to multiply to a level 102- to 103-fold lower than that of the wild-type strain. This is the first comprehensive report of the genetic and transcriptional organization of the hrp gene cluster in a phytopathogenic bacterium.

L5 ANSWER 12 OF 14 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 7
AN 1992:131656 BIOSIS
DN BR42:59356
TI GENES AND SIGNALS CONTROLLING THE PSEUDOMONAS-SYRINGAE PV. PHASEOLICOLA AND PLANT INTERACTION.
AU FELLAY R; ***RAHME L G*** ; MINDRINOS M N; FREDERICK R D; PISI A; PANOPOULOS N J
CS DEP. PLANT PATHOL., UNIV. CALIF., BERKELEY, CALIF. 94729, USA.
SO HENNECKE, H. AND D. P. S. VERMA (ED.). CURRENT PLANT SCIENCE AND BIOTECHNOLOGY IN AGRICULTURE, VOL. 10. ADVANCES IN MOLECULAR GENETICS OF PLANT-MICROBE INTERACTIONS, VOL. 1; 5TH INTERNATIONAL SYMPOSIUM ON THE MOLECULAR GENETICS OF PLANT-MICROBE INTERACTIONS,
INTERLAKEN, SWITZERLAND, SEPTEMBER 9-14, 1990. XV+482P. KLUWER ACADEMIC PUBLISHERS: DORDRECHT, NETHERLANDS; NORWELL, MASSACHUSETTS,
USA. ILLUS. (1991) 0 (0), 45-52.
ISBN: 0-7923-1082-9.
DT Conference
FS BR; OLD
LA English

L5 ANSWER 13 OF 14 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 8
AN 1990:364660 BIOSIS
DN BR39:49136
TI STRUCTURE FUNCTION REGULATION AND EVOLUTION OF GENES INVOLVED IN
PATHOGENICITY THE HYPERSENSITIVE RESPONSE AND PHASEOLOTOXIN IMMUNITY
IN THE BEAN HALO BLIGHT PATHOGEN.
AU MINDRINOS M N; ***RAHME L G*** ; FREDERICK R D; HATZILOUKAS E; GRIMM C; PANOPOULOS N J
CS DEP. PLANT PATHOL., UNIV. CALIF., BERKELEY, CALIF. 94720.

SO SILVER, S., ET AL. (ED.). PSEUDOMONAS: BIOTRANSFORMATIONS, PATHOGENESIS, AND EVOLVING BIOTECHNOLOGY; SYMPOSIUM, CHICAGO, ILLINOIS, USA, JULY 1989. XXIV+423P. AMERICAN SOCIETY FOR MICROBIOLOGY: WASHINGTON, D.C., USA. ILLUS. (1990) 0 (0), 74-81.
ISBN: 1-55581-019-5.
DT Conference
FS BR; OLD
LA English

L5 ANSWER 14 OF 14 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 9
AN 1989:303713 BIOSIS
DN BR37:18090
TI THE COMMON PATHOGENICITY GENES OF PSEUDOMONAS-SYRINGAE PATHOVARS.
AU GRIMM C; ***RAHME L*** ; FREDERICK R; MINDRINOS M; LINDGREN P B; PANOPOULOS N J
CS DEP. PLANT PATHOL., UNIV. CALIF., BERKELEY, CALIF. 94720.
SO STASKAWICZ, B., P. AHLQUIST AND O. YODER (ED.). UCLA (UNIVERSITY OF CALIFORNIA-LOS ANGELES) SYMPOSIA ON MOLECULAR AND CELLULAR BIOLOGY
NEW SERIES, VOL. 101. MOLECULAR BIOLOGY OF PLANT-PATHOGEN INTERACTIONS; STEAMBOAT SPRINGS, COLORADO, USA, MARCH 26-APRIL 1, 1988. XVIII+307P. ALAN R. LISS, INC.: NEW YORK, NEW YORK, USA.
ILLUS. (1989) 0 (0), 49-56.
CODEN: USMBD6. ISSN: 0735-9543. ISBN: 0-8451-4700-5.
FS BR; OLD
LA English

=> e tan man/au

E1 12 TAN M Z/AU
E2 4 TAN MABEL H J/AU
E3 0 --> TAN MAN/AU
E4 1 TAN MAN CHI/AU
E5 2 TAN MAN QI/AU
E6 6 TAN MAN WAH/AU
E7 4 TAN MANDY M/AU
E8 7 TAN MANQI/AU
E9 5 TAN MANQING/AU
E10 1 TAN MANQIO/AU
E11 5 TAN MANQIU/AU
E12 1 TAN MANQU/AU
=> s e6
L6 6 "TAN MAN WAH"/AU
=> dup rem l6

L7 4 DUP REM L6 (2 DUPLICATES REMOVED)
=> d bib ab 1-

YOU HAVE REQUESTED DATA FROM 4 ANSWERS - CONTINUE? Y/(N):y

L7 ANSWER 1 OF 4 CAPLUS COPYRIGHT 1998 ACS
AN 1998:744978 CAPLUS
DN 130:2014
TI Methods of screening compounds useful for prevention of infection or pathogenicity
IN Ausubel, Frederick M.; Rahme, Lawrence G.; ***Tan, Man-wah*** ; Ruvkun, Gary B.; Mahajan-Miklos, Shalina; Broeks, Annegien; Plasterk, Ronald H. A.; Jander, Georg; Heard, Jacqueline
PA The General Hospital Corp., USA; The Netherlands Cancer Institute
SO PCT Int. Appl., 87 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9850080	A1	19981112	WO 98-US9150	19980508

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

PRAI US 97-852927 19970508
US 97-962750 19971103
AB Screening procedures are disclosed for identifying compds. useful for inhibiting infection or pathogenicity. Methods are also disclosed for identifying pathogenic virulence factors.

L7 ANSWER 2 OF 4 CAPLUS COPYRIGHT 1998 ACS
AN 1997:668606 CAPLUS
DN 127:260044
TI Genetic analysis of virulence in the transkingdom pathogen, Pseudomonas aeruginosa, and of host responses in Caenorhabditis elegans (pyocyanin)
AU ***Tan, Man-Wah***

CS Harvard Univ., Cambridge, MA, USA
SO (1997) 213 pp. Avail.: UMI, Order No. DA9733197
From: Diss. Abstr. Int., B 1997, 58(5), 2262
DT Dissertation
LA English
AB Unavailable

L7 ANSWER 3 OF 4 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 1
AN 1998:49050 BIOSIS
DN PREV199800049050
TI Use of model plant hosts to identify Pseudomonas aeruginosa virulence factors.
AU Rahme, Laurence G.; ***Tan, Man-Wah*** ; Le, Long; Wong, Sandy M.; Tompkins, Ronald G.; Calderwood, Stephen B.; Ausubel, Frederick M. (1)
CS (1) Dep. Genetics Harvard Med. Sch., Dep. Molecular Biol., Massachusetts General Hosp., Boston, MA 02114 USA
SO Proceedings of the National Academy of Sciences of the United States of America, (Nov. 25, 1997) Vol. 94, No. 24, pp. 13245-13250. ISSN: 0027-8424.
DT Article
LA English
AB We used plants as an in vivo pathogenesis model for the identification of virulence factors of the human opportunistic pathogen Pseudomonas aeruginosa. Nine of nine TnphoA mutant derivatives of P. aeruginosa strain UCBPPPA14 that were identified in a plant leaf assay for less pathogenic mutants also exhibited significantly reduced pathogenicity in a burned mouse pathogenicity model, suggesting that P. aeruginosa utilizes common strategies to infect both hosts. Seven of these nine mutants contain TnphoA insertions in previously unknown genes. These results demonstrate that an alternative nonvertebrate host of a human bacterial pathogen can be used in an in vivo high throughput screen to identify novel bacterial virulence factors involved in mammalian pathogenesis.

L7 ANSWER 4 OF 4 CAPLUS COPYRIGHT 1998 ACS
AN 1996:679267 CAPLUS
DN 125:317307
TI Bioassay methods using different eukaryotic organisms for screening compounds useful for prevention of infection or pathogenicity and for identifying pathogenic virulence factors
IN Ausubel, Frederick M.; Rahme, Laurence G.; ***Tan, Man-Wah*** ; Ruvkun, Gary B.
PA General Hospital Corporation, USA
SO PCT Int. Appl., 50 pp.

CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9630053	A1	19961003	WO 96-US4210	19960327
W: CA, JP				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
CA 2215836	AA	19961003	CA 96-2215836	19960327
EP 828520	A1	19980318	EP 96-911451	19960327
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				

PRAI US 95-411560 19950328
WO 96-US4210 19960327

AB Screening procedures are disclosed for identifying compds. useful for inhibiting infection or pathogenicity. The methodol. of the invention involves (a) exposing .gtoreq.2 different eukaryotic organisms, .gtoreq.1 of which is a non-rodent, to a single pathogen in the presence of .gtoreq.1 candidate compd; and (b) identifying a compd. that inhibits the pathogen in each of the eukaryotic organisms. Methods are also disclosed for identifying pathogenic virulence factors.

=> e ruvkun gary/au

E1	2	RUVKUN G */AU
E2	47	RUVKUN G B/AU
E3	56	--> RUVKUN GARY/AU
E4	9	RUVKUN GARY B/AU
E5	1	RUVKUN GARY BRUCE/AU
E6	1	RUVKUN S/AU
E7	20	RUVO A D/AU
E8	1	RUVO A DE/AU
E9	4	RUVO L/AU
E10	23	RUVO M/AU
E11	20	RUVO MENOTTI/AU
E12	19	RUVOEN CLOUET N/AU

=> s e1-e5

L8 115 ("RUVKUN G *"/AU OR "RUVKUN G B"/AU OR "RUVKUN GARY"/AU OR "RUVKUN GARY B"/AU OR "RUVKUN GARY BRUCE"/AU)

=> dup rem l8

PROCESSING COMPLETED FOR L8
L9 65 DUP REM L8 (50 DUPLICATES REMOVED)

=> s l9 and ((pathogen?) or (inhibit?))

2 FILES SEARCHED...
4 FILES SEARCHED...
6 FILES SEARCHED...
L10 4 L9 AND ((PATHOGEN?) OR (INHIBIT?))

=> d bib ab 1-

YOU HAVE REQUESTED DATA FROM 4 ANSWERS - CONTINUE? Y/(N):y

L10 ANSWER 1 OF 4 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1998:433064 BIOSIS
DN PREV199800433064
TI Caenorhabditis elegans Akt/PKB transduces insulin receptor-like signals from AGE-1 PI3 kinase to the DAF-6 transcription factor.
AU Paradis, Suzanne; ***Ruvkun, Gary (1)***
CS (1) Dep. Genet., Mass. Gen. Hosp., Harv. Med. Sch., Boston, MA 02114 USA
SO Genes & Development, (Aug. 15, 1998) Vol. 12, No. 16, pp. 2488-2498. ISSN: 0890-9369.
DT Article
LA English
AB A neurosecretory pathway regulates a reversible developmental arrest and metabolic shift at the Caenorhabditis elegans dauer larval stage. Defects in an insulin-like signaling pathway cause arrest at the dauer.stage. We show here that two C. elegans Akt/PKB homologs, akt-1 and akt-2, transduce insulin receptor-like signals that ***inhibit*** dauer arrest and that AKT-1 and AKT-2 signaling are indispensable for insulin receptor-like signaling in C. elegans. A loss-of-function mutation in the Fork head transcription factor DAF-16 relieves the requirement for Akt/PKB signaling, which indicates that AKT-1 and AKT-2 function primarily to antagonize DAF-16. This is the first evidence that the major target of Akt/PKB signaling is a of wild-type akt-1 relieves the requirement for signaling from AGE-1 PI3K, which acts downstream of the transcription factor. An activating mutation in akt-1, revealed by a genetic screen, as well as increased dosage of wild-type akt-1 relieves the requirement for signaling from AGE-1 P13K, which acts

downstream of the DAF-2 insulin/IGF-1 receptor homolog. This demonstrates that Akt/PKB activity is not necessarily dependent on AGE-1 P13K activity, akt-1 and akt-2 are expressed in overlapping patterns in the nervous system and in tissues that are remodeled during dauer formation.

L10 ANSWER 2 OF 4 CAPLUS COPYRIGHT 1998 ACS
AN 1998:744978 CAPLUS
DN 130:2014
TI Methods of screening compounds useful for prevention of infection or ***pathogenicity***
IN Ausubel, Frederick M.; Rahme, Lawrence G.; Tan, Man-wah; ***Ruvkun, Gary B.*** ; Mahajan-Miklos, Shalina; Broeks, Annegien; Plasterk, Ronald H. A.; Jander, Georg; Heard, Jacqueline
PA The General Hospital Corp., USA; The Netherlands Cancer Institute
SO PCT Int. Appl., 87 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9850080	A1	19981112	WO 98-US9150	19980508

PI W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG
PRAI US 97-852927 19970508
US 97-962750 19971103
AB Screening procedures are disclosed for identifying compds. useful for ***inhibiting*** infection or ***pathogenicity*** . Methods are also disclosed for identifying ***pathogenic*** virulence factors.

L10 ANSWER 3 OF 4 CAPLUS COPYRIGHT 1998 ACS
AN 1996:679267 CAPLUS
DN 125:317307
TI Bioassay methods using different eukaryotic organisms for screening compounds useful for prevention of infection or ***pathogenicity*** and for identifying ***pathogenic***

virulence factors
IN Ausubel, Frederick M.; Rahme, Laurence G.; Tan, Man-Wah; ***Ruvkun, Gary B.***
PA General Hospital Corporation, USA
SO PCT Int. Appl., 50 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9630053	A1	19961003	WO 96-US4210	19960327
W: CA, JP				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
CA 2215836	AA	19961003	CA 96-2215836	19960327
EP 828520	A1	19980318	EP 96-911451	19960327
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				

PRAI US 95-411560 19950328
WO 96-US4210 19960327
AB Screening procedures are disclosed for identifying compds. useful for ***inhibiting*** infection or ***pathogenicity*** . The methodol. of the invention involves (a) exposing .gtoreq.2 different eukaryotic organisms, .gtoreq.1 of which is a non-rodent, to a single ***pathogen*** in the presence of .gtoreq.1 candidate compd; and (b) identifying a compd. that ***inhibits*** the ***pathogen*** in each of the eukaryotic organisms. Methods are also disclosed for identifying ***pathogenic*** virulence factors.

L10 ANSWER 4 OF 4 CAPLUS COPYRIGHT 1998 ACS
AN 1984:566961 CAPLUS
DN 101:166961
TI Genetic analysis of symbiotic nitrogen fixation
AU Long, S. R.; ***Ruvkun, G. B.*** ; Meade, H. M.; Buikema, W. E.; Brown, S. E.; Friedman, A. M.; Ausubel, F. M.
CS Dep. Biol. Sci., Stanford Univ., Stanford, CA, 94305, USA
SO Dev. Ind. Microbiol. (1983), 24, 21-9
CODEN: DIMCAL; ISSN: 0070-4563
DT Journal
LA English
AB Mutants are useful in the study of how genes control complex biol. processes. Transposon mutagenesis and recombinant DNA techniques were used to generate and analyze symbiotic mutants of Rhizobium

meliloti, a bacterium which forms N2-fixing root nodules in assocn. with a host plant, alfalfa. A wild-type strain of R. meliloti was treated with a transposon-carrying suicide plasmid. Resulting colonies which contained a transposon were tested on plants for symbiotic properties. .apprx.20 Tn5-caused symbiotic mutants were isolated. Some of these resulted from concerted transporition of both phage Mu and Tn5. In addn., some mutants were caused by insertion of a native R. meliloti insertion sequence. The symbiotic mutants obtained were of several types. Some did not invade host root hairs or stimulate nodule formation. Others formed nodules and invaded host cells, but did not differentiate or fix N2. To study the expression of the genes controlling the symbiosis, the genes identified by transposon insertion were cloned. A general procedure was also developed for cloning infection and nodulation genes by complementation of nonnodulating mutants. This procedure uses the host plant to select functionally complemented mutants from a population of mutants contg. a random clone bank. It utilizes a broad host range conjugatable cosmid vector which accepts large inserts and transfers DNA at high efficiency into recipient Escherichia coli cells. This strategy should be applicable to spontaneous or chem. induced mutants, and may be useful in cloning infection genes of ***pathogens*** .

=> e mahajanmiklos shalina/au

E1 9 MAHAJANI VIJAYKUMAR V/AU
E2 3 MAHAJANMIKLOS S/AU
E3 0 --> MAHAJANMIKLOS SHALINA/AU
E4 1 MAHAJANS CS/AU
E5 1 MAHAJANS S K/AU
E6 1 MAHAJANSHETTI C C/AU
E7 70 MAHAJANSHETTI C S/AU
E8 1 MAHAJANSHETTI CHANABASAPPA S/AU
E9 6 MAHAJANSHETTI CHANBASAPPA S/AU
E10 2 MAHAJANSHETTI CHENNABASAPPA S/AU
E11 1 MAHAJANSHETTI S B/AU
E12 3 MAHAJANSHETTY C S/AU
=> s e2
L11 3 "MAHAJANMIKLOS S"/AU
=> dup rem l11
L12 3 DUP REM L11 (0 DUPLICATES REMOVED)
=> d bib ab 1-
YOU HAVE REQUESTED DATA FROM 3 ANSWERS - CONTINUE? Y/(N):y

L12 ANSWER 1 OF 3 SCISEARCH COPYRIGHT 1998 ISI (R)
AN 1998:169094 SCISEARCH
GA The Genuine Article (R) Number: YY174
TI Drosophila fascin mutants are rescued by overexpression of the villin-like protein, quail
AU Cant K; Knowles B A; ***MahajanMiklos S*** ; Heintzelman M; Cooley L (Reprint)
CS YALE UNIV, SCH MED, DEPT GENET, 333 CEDAR ST, NEW HAVEN, CT 06510 (Reprint); YALE UNIV, SCH MED, DEPT GENET, NEW HAVEN, CT 06510; DARTMOUTH COLL SCH MED, DEPT ANAT, HANOVER, NH 03755; DARTMOUTH COLL SCH MED, DEPT PATHOL, HANOVER, NH 03755
CYA USA
SO JOURNAL OF CELL SCIENCE, (JAN 1998) Vol. 111, Part 2, pp. 213-221. Publisher: COMPANY OF BIOLOGISTS LTD, BIDDER BUILDING CAMBRIDGE COMMERCIAL PARK COWLEY RD, CAMBRIDGE, CAMBS, ENGLAND CB4 4DL. ISSN: 0021-9533.
DT Article; Journal
FS LIFE
LA English
REC Reference Count: 53
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Actin bundle assembly in specialized structures such as microvilli on intestinal epithelia and Drosophila bristles requires two actin bundling proteins. In these systems, the distinct biochemical properties and temporal localization of actin bundling proteins suggest that these proteins are not redundant, During Drosophila oogenesis, the formation of cytoplasmic actin bundles in nurse cells requires two actin bundling proteins, fascin encoded by the singed gene and a villin-like protein encoded by the quail gene, singed and quail mutations are fully recessive and each mutation disrupts nurse cell cytoplasmic actin bundle formation, We used P-element mediated germline transformation to overexpress quail in singed mutants and test whether these proteins have redundant functions in vivo, Overexpression of quail protein in a sterile singed background restores actin bundle formation in egg chambers, The degree of rescue by quail depends on the level of quail protein overexpression, as well as residual levels of fascin function, In nurse cells that contain excess quail but no fascin, the cytoplasmic actin network initially appears wild type hut then becomes disorganized in the final stages of nurse cell cytoplasm transport, The ability of quail overexpression to compensate for the absence of fascin demonstrates that fascin is partially redundant with quail in the Drosophila germline, Quail appears to function as a bundle

initiator while fascin provides bundle organization.

L12 ANSWER 2 OF 3 SCISEARCH COPYRIGHT 1998 ISI (R)
AN 94:708500 SCISEARCH
GA The Genuine Article (R) Number: PP811
TI INTERCELLULAR CYTOPLASM TRANSPORT DURING DROSOPHILA OOGENESIS
AU ***MAHAJANMIKLOS S (Reprint)*** ; COOLEY L
CS YALE UNIV, SCH MED, DEPT GENET, 333 CEDAR ST, NEW HAVEN, CT, 06510 (Reprint)
CYA USA
SO DEVELOPMENTAL BIOLOGY, (OCT 1994) Vol. 165, No. 2, pp. 336-351.
ISSN: 0012-1606.
DT General Review; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 122

L12 ANSWER 3 OF 3 SCISEARCH COPYRIGHT 1998 ISI (R)
AN 94:451398 SCISEARCH
GA The Genuine Article (R) Number: NZ242
TI THE VILLIN-LIKE PROTEIN ENCODED BY THE DROSOPHILA QUAIL GENE IS REQUIRED FOR ACTIN BUNDLE ASSEMBLY DURING OOGENESIS
AU ***MAHAJANMIKLOS S (Reprint)*** ; COOLEY L
CS YALE UNIV, SCH MED, DEPT GENET, NEW HAVEN, CT, 06510 (Reprint)
CYA USA
SO CELL, (29 JUL 1994) Vol. 78, No. 2, pp. 291-301.
ISSN: 0092-8674.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 54
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Mutations in the Drosophila quail gene result in female sterility due to the disruption of cytoplasmic transport from the nurse cells into the oocyte late in oogenesis. Nurse cells from quail mutant egg chambers fail to assemble cytoplasmic actin filament bundles correctly. We have cloned the quail gene and found that it encodes a protein with homology to the vertebrate actin-regulating protein villin. Unlike vertebrate villin, which is restricted to specialized absorptive epithelial cells, the villin-like protein encoded by quail is germline specific in adult flies. Antibodies directed against the quail protein show a striking colocalization with filamentous actin in the nurse cells and the oocyte. Our results demonstrate that the villin-like product of quail is required for the formation of cytoplasmic actin filament bundles in nurse cells,

possibly by regulating both the polymerization and organization of actin filaments as demonstrated for vertebrate villin in vitro.

=> e broeks annegien/au

E1 26 BROEKS A/AU
E2 1 BROEKS A G M/AU
E3 9 --> BROEKS ANNEGIEN/AU
E4 1 BROEKS M W A/AU
E5 1 BROEKSEMA EGBERT/AU
E6 2 BROEKSEMA R/AU
E7 1 BROEKSEMA SLOT F J/AU
E8 1 BROEKSEMASLOT F J/AU
E9 1 BROEKSMA A/AU
E10 2 BROEKSMA A H/AU
E11 13 BROEKSMA C/AU
E12 1 BROEKSMA CARYN/AU

=> s e1-e3

L13 36 ("BROEKS A"/AU OR "BROEKS A G M"/AU OR "BROEKS ANNEGIEN"/AU)

=> dup rem l13

PROCESSING COMPLETED FOR L13
L14 10 DUP REM L13 (26 DUPLICATES REMOVED)

=> d bib ab 1-

YOU HAVE REQUESTED DATA FROM 10 ANSWERS - CONTINUE? Y/(N):y

L14 ANSWER 1 OF 10 CAPLUS COPYRIGHT 1998 ACS
AN 1998:744978 CAPLUS
DN 130:2014
TI Methods of screening compounds useful for prevention of infection or pathogenicity
IN Ausubel, Frederick M.; Rahme, Lawrence G.; Tan, Man-wah; Ruvkun, Gary B.; Mahajan-Miklos, Shalina; ***Broeks, Annegien*** ; Plasterk, Ronald H. A.; Jander, Georg; Heard, Jacqueline
PA The General Hospital Corp., USA; The Netherlands Cancer Institute
SO PCT Int. Appl., 87 pp.
CODEN: PIXXD2
DT Patent

LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9850080	A1	19981112	WO 98-US9150	19980508

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

PRAI US 97-852927 19970508
US 97-962750 19971103

AB Screening procedures are disclosed for identifying compds. useful for inhibiting infection or pathogenicity. Methods are also disclosed for identifying pathogenic virulence factors.

L14 ANSWER 2 OF 10 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 1
AN 1998:496117 BIOSIS
DN PREV199800496117
TI ATM germline mutations in classical ataxia-telangiectasia patients in the Dutch population.
AU ***Broeks, A.*** ; De Klein, A.; Floore, A. N.; Muijtjens, M.; Kleijer, W. J.; Jaspers, N. G.; Van't Veer, L. J. (1)
CS (1) Netherlands Cancer Inst., Plesmanlaan 121, 1066 CX Amsterdam Netherlands
SO Human Mutation, (1998) Vol. 12, No. 5, pp. 330-337.
ISSN: 1059-7794.
DT Article
LA English
AB Germline mutations in the ATM gene are responsible for the autosomal recessive disorder ataxia-telangiectasia (A-T). In our study, we have determined the ATM mutation spectrum in 19 classical A-T patients, including some immigrant populations, as well as 12 of Dutch ethnic origin. Both the protein truncation test (PTT) and the restriction endonuclease fingerprinting (REF) method were used and compared for their detection efficiency, identifying 76% and 60% of the mutations, respectively. Most patients were found to be compound heterozygote. Seventeen mutations were distinct, of which 10 were not reported previously. Mutations are small deletions or point mutations frequently affecting splice sites. Moreover, a 16.7-kb genomic deletion of the 3' end of the gene, most likely a

result of recombination between two LINE elements, was identified. The most frequently found mutation, identified in three unrelated Turkish A-T individuals, was previously described to be a Turkish A-T founder mutation. The presence of a founder mutation among relatively small ethnic population groups in Western Europe could indicate a high carrier frequency in such communities. In patients of Dutch ethnic origin, however, no significant founder effect could be identified. The observed genetic heterogeneity including the relative high percentage of splice-site mutations had no reflection on the phenotype. All patients manifested classical A-T and increased cellular radioresistant DNA synthesis.

L14 ANSWER 3 OF 10 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1998:195111 BIOSIS
DN PREV199800195111
TI Breast cancer susceptibility: ATM germline mutations and radiation exposure.
AU Van't Veer, L. J. (1); ***Broeks, A.*** ; Floore, A. N.; Urbanus, J. H. M.; Dahler, E.; Russell, N. S.; Hogervorst, F. B. L.; Van Leeuwen, F. E.
CS (1) Dep. Pathol., Netherlands Cancer Inst., Amsterdam Netherlands
SO Proceedings of the American Association for Cancer Research Annual Meeting, (March, 1998) Vol. 39, pp. 181.
Meeting Info.: 89th Annual Meeting of the American Association for Cancer Research New Orleans, Louisiana, USA March 28-April 1, 1998 American Association for Cancer Research
. ISSN: 0197-016X.
DT Conference
LA English

L14 ANSWER 4 OF 10 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 2
AN 1997:19203 BIOSIS
DN PREV199799318406
TI Homologues of the human multidrug resistance genes MRP and MDR contribute to heavy metal resistance in the soil nematode Caenorhabditis elegans.
AU ***Broeks, Annegien*** ; Gerrard, Bernard; Allikmets, Rando; Dean, Michael; Plasterk, Ronald H. A. (1)
CS (1) Div. Molecular Biol., Neth. Cancer Inst., Plesmanlaan 121, 1066 CX Amsterdam Netherlands
SO EMBO (European Molecular Biology Organization) Journal, (1996) Vol. 15, No. 22, pp. 6132-6143.
ISSN: 0261-4189.
DT Article
LA English

AB Acquired resistance of mammalian cells to multiple chemotherapeutic drugs can result from enhanced expression of the multidrug resistance-associated protein (MR.P), which belongs to the ABC transporter superfamily. ABC transporters play a role in the protection of organisms against exogenous toxins by cellular detoxification processes. We have identified four MRP homologues in the soil nematode Caenorhabditis elegans, and we have studied one member, mrp-1, in detail. Using an mrp::lacZ gene fusion, mrp-1 expression was found in cells of the pharynx, the pharynx-intestinal valve and the anterior intestinal cells, the rectum-intestinal valve and the epithelial cells of the vulva. Targeted inactivation of mrp-1 resulted in increased sensitivity to the heavy metal ions cadmium and arsenite, to which wild-type worms are highly tolerant. The most pronounced effect of the mrp-1 mutation is on the ability of animals to recover from temporary exposure to high concentrations of heavy metals. Nematodes were found to be hypersensitive to heavy metals when both the MRP homologue, mrp-1, and a member of the P-glycoprotein (Pgp) gene family pgp-1, were deleted. We conclude that nematodes have multiple proteins, homologues of mammalian proteins involved in the cellular resistance to chemotherapeutic drugs, that protect them against heavy metals.

L14 ANSWER 5 OF 10 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 3
AN 1995:307591 BIOSIS
DN PREV199598321891
TI A P-glycoprotein protects Caenorhabditis elegans against natural toxins.
AU ***Broeks, Annegien*** ; Janssen, Hans W. R. M.; Calafat, Jero; Plasterk, Ronald H. A. (1)
CS (1) Div. Molecular Biol., Netherland Cancer Inst., Plesmanlaan 121, 1066 CX Amsterdam Netherlands
SO EMBO (European Molecular Biology Organization) Journal, (1995) Vol. 14, No. 9, pp. 1858-1866.
ISSN: 0261-4189.
DT Article
LA English
AB P-glycoproteins can cause resistance of mammalian tumor cells to chemotherapeutic drugs. They belong to an evolutionarily well-conserved family of ATP binding membrane transporters. Four P-glycoprotein gene homologs have been found in the nematode Caenorhabditis elegans; this report describes the functional analysis of two. We found that PGP-3 is expressed in both the apical membrane of the excretory cell and in the apical membrane of intestinal cells, whereas PGP-1 is expressed only in the apical membrane of the intestinal cells and the intestinal valve. By

transposon-mediated deletion mutagenesis we generated nematode strains with deleted P-glycoprotein genes and found that the pgp-3 deletion mutant, but not the pgp-1 mutant, is sensitive to both colchicine and chloroquine. Our results suggest that soil nematodes have P-glycoproteins to protect themselves against toxic compounds made by plants and microbes in the rhizosphere.

L14 ANSWER 6 OF 10 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 4
AN 1993:477773 BIOSIS
DN PREV199396111373
TI Target-selected gene inactivation in Caenorhabditis elegans by using a frozen transposon insertion mutant bank.
AU Zwaal, Richard R.; ***Broeks, Annegien*** ; Van Meurs, Joyce; Groenen, Jose T. M.; Plasterk, Ronald H. A. (1)
CS (1) Div. Mol. Biol., Netherlands Cancer Inst., Plesmanlaan 121, 1066 CX Amsterdam, The Netherlands
SO Proceedings of the National Academy of Sciences of the United States of America, (1993) Vol. 90, No. 16, pp. 7431-7435.
ISSN: 0027-8424.
DT Article
LA English
AB To understand how genotype determines the phenotype of the animal Caenorhabditis elegans, one ideally needs to know the complete sequence of the genome and the contribution of genes to phenotype, which requires an efficient strategy for reverse genetics. We here report that the Tc1 transposon induces frequent deletions of flanking DNA, apparently resulting from Tc1 excision followed by imprecise DNA repair. We use this to inactivate genes in two steps, (i) We established a frozen library of 5000 nematode lines mutagenized by Tc1 insertion, from which insertion mutants of genes of interest can be recovered. Their address within the library is determined by PCR. (ii) Animals are then screened, again by PCR, to detect derivatives in which Tc1 and 1000-2000 base pairs of Ranking DNA are deleted, and thus a gene of interest is inactivated. We have thus far isolated Tc1 insertions in 16 different genes and obtained deletion derivatives of 6 of those.

L14 ANSWER 7 OF 10 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 5
AN 1993:274193 BIOSIS
DN PREV199396004418
TI The expression of two P-glycoprotein (pgp) genes in transgenic Caenorhabditis elegans is confined to intestinal cells.
AU Lincke, Carsten R. (1); ***Broeks, Annegien*** ; The, Inge; Plasterk, Ronald H. A.; Borst, Piet
CS (1) Dep. Pediatrics, Acad. Med. Cent., Univ. Amsterdam, Meibergdreef

9, 1105 AZ Amsterdam Netherlands
SO EMBO (European Molecular Biology Organization) Journal, (1993) Vol. 12, No. 4, pp. 1615-1620.
ISSN: 0261-4189.
DT Article
LA English
AB P-glycoproteins can cause multidrug resistance in mammalian tumor cells by active extrusion of cytotoxic drugs. The natural function of these evolutionarily conserved, membrane-bound ATP binding transport proteins is unknown. In mammals, P-glycoproteins are abundantly present in organs associated with the digestive tract. We have studied the tissue-specific expression of Caenorhabditis elegans P-glycoprotein genes pgp-1 and pgp-3 by transformation of nematodes with pgp-lacZ gene fusion constructs in which the promoter area of the pgp genes was fused to the coding region of lacZ. Expression of pgp-1 and pgp-3, as inferred from pgp-lacZ transgenic nematodes, was confined to the intestinal cells. The expression patterns of both genes were virtually indistinguishable. Quantitative analysis of pgp mRNA levels during development showed that pgp-1, -2, and -3 were expressed throughout the life cycle of C. elegans, albeit with some variation indicating developmental regulation. The expression of P-glycoprotein genes in intestinal cells is an evolutionarily conserved feature of these genes, consistent with the hypothesis that P-glycoproteins provide a mechanism of protection against environmental toxins.

L14 ANSWER 8 OF 10 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1993:219844 BIOSIS
DN PREV199344104344
TI Reverse genetics of Caenorhabditis elegans, using the Tc1 transposon.
AU Plasterk, Ronald H. A.; Groenen, Jose T. M.; Van Meurs, Joyce; ***Broeks, A.*** ; Zwaal, R.; Youngman, S.
CS Dep. Molecular Biol., Netherlands Cancer Institute, Plesmanlaan 121, 1066CX Amsterdam Netherlands
SO Journal of Cellular Biochemistry Supplement, (1993) Vol. 0, No. 17 PART C, pp. 117.
Meeting Info.: Keystone Symposium on Molecular Helminthology: An Integrated Approach Tamarron, Colorado, USA February 10-17, 1993
ISSN: 0733-1959.
DT Conference
LA English

L14 ANSWER 9 OF 10 SCISEARCH COPYRIGHT 1998 ISI (R)
AN 93:127771 SCISEARCH

GA The Genuine Article (R) Number: KN466
TI REVERSE GENETICS OF C-ELEGANS, USING THE TC1 TRANSPOSON
AU PLASTERK R H A (Reprint); GROENEN J T M; VANMEURS J; ***BROEKS*** *** A*** ; ZWAAL R; YOUNGMAN S
CS NETHERLANDS CANC INST, DEPT MOLEC BIOL, 1066 CX AMSTERDAM, NETHERLANDS
CYA NETHERLANDS
SO JOURNAL OF CELLULAR BIOCHEMISTRY, (08 FEB 1993) Supp. 17C, pp. 117. ISSN: 0730-2312.
DT Conference; Journal
FS LIFE
LA ENGLISH
REC No References

L14 ANSWER 10 OF 10 CABA COPYRIGHT 1998 CABI
AN 92:55808 CABA
DN 921895309
TI Turnover and growth: the end? - A study in collaboration with the Accountancy and Tax Advice Firm NCB
Omzet & aanwas: het einde? - Een onderzoek in samenwerking met Accountants- en Belastingadviesbureau NCB
AU ***Broeks, A. G. M.*** ; Poppe, K. J.; Beek, P. C. M. van; Van Beek, P. C. M.
SO Mededelingen - Landbouw-Economisch Instituut, (1991) No. 448, pp. 36. 4 fig. 10 ref.The Hague
Price: Dfl 11.
ISBN: 90-5242-127-7
CY Netherlands Antilles
DT Miscellaneous
LA Dutch
AB In profit and loss accounts and management systems the item 'turnover and growth' occurs and frequently causes difficulties of interpretation. In the Netherlands there is an increasing need for an understanding of the detailed costs of cattle holdings. With the aid of the theory of profit and loss accounting it is shown that this understanding is improved by modifying the design of the profit and loss account. A distinction is made between breeding and productive cattle (fixed assets, means of production) on the one hand and fattening and trade cattle on the other (liquid assets, product being processed). The concept of turnover and growth is then no longer needed.

=> e plasterk donald/au

E1 4 PLASTERER T N/AU

E2 8 PLASTERER THOMAS N/AU
E3 0 --> PLASTERK DONALD/AU
E4 4 PLASTERK K J/AU
E5 22 PLASTERK R/AU
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E8 272 PLASTERK R H A/AU
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E12 2 PLASTERK RONALD H/AU

=> e plasterk ronald/au

E1 14 PLASTERK R H A */AU
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E4 2 PLASTERK RONALD H/AU
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E6 1 PLASTERK RONALD HANS ANTON/AU
E7 1 PLASTERKIRK L E/AU
E8 1 PLASTIC STEJIC M/AU
E9 1 PLASTICS DOW/AU
E10 1 PLASTICS EDITORIAL STAFF/AU
E11 1 PLASTICS ENGINEERING CO/AU
E12 1 PLASTICUS I/AU

=> s e1-e6

L15 155 ("PLASTERK R H A *"/AU OR "PLASTERK ROANLD H A"/AU OR "PLASTERK RONALD"/AU OR "PLASTERK RONALD H"/AU OR "PLASTER K RONALD H A"/AU OR "PLASTERK RONALD HANS ANTON"/AU)

=> dup rem l15

PROCESSING COMPLETED FOR L15
L16 93 DUP REM L15 (62 DUPLICATES REMOVED)

=> s l16 and ((pathogen?) or (inhibit?))

2 FILES SEARCHED...
4 FILES SEARCHED...
6 FILES SEARCHED...
L17 6 L16 AND ((PATHOGEN?) OR (INHIBIT?))

=> d bib ab 1-

YOU HAVE REQUESTED DATA FROM 6 ANSWERS - CONTINUE? Y/(N):y

L17 ANSWER 1 OF 6 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1998:210148 BIOSIS
DN PREV199800210148
TI HIV integrase: A target for drug discovery.
AU Lutzke, Ramon A. Puras; ***Plasterk, Ronald H. A. (1)***
CS (1) Dep. Biochemistry Molecular Biology, Free University Amsterdam, De Boelelaan 1084, HV Amsterdam Netherlands
SO Genes and Function, (Dec., 1997) Vol. 1, No. 5-6, pp. 289-307. ISSN: 1360-7413.
DT General Review
LA English
AB Current antiviral strategies against HIV rely on structure-function analysis of HIV reverse transcriptase (RT) and protease (PR). The third viral pol gene product, HIV integrase (IN), is also a good target for drug discovery, since IN is essential for retroviral replication and, moreover, it has no obvious functional analogue in the host. IN forms a ternary complex with metal ions and DNA and has a mechanism of catalysis common with other polynucleotidyl transferases. Although there is no structural information for full-length IN available, structures of all three functional IN domains have been determined by X-ray crystallography and NMR spectroscopy. The N-terminal domain has a novel zinc-binding fold, the catalytic domain shares a common structural motif with other polynucleotidyl transferases, and the C-terminal DNA-binding domain has a Src-homology-3-like fold. This structural information provides the basis for drug development. In rum, increasing numbers of IN ***inhibitors*** identified so far may serve structure-function analysis of IN. The final goal is the development of new classes of anti-HIV drugs, which can be added to the repertoire of anti-RT and anti-PR drugs.

L17 ANSWER 2 OF 6 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1996:56959 BIOSIS
DN PREV199698629094
TI Identification of a hexapeptide ***inhibitor*** of the human immunodeficiency virus integrase protein by using a combinatorial chemical library.
AU Puras Lutzke, Ramon A.; Eppens, Noor A.; Weber, Patricia A.; Houghten, Richard A.; ***Plasterk, Ronald H. A. (1)***
CS (1) Div. Mol. Biol., Netherlands Cancer Inst., Plesmanlaan 121, 1066

CX Amsterdam Netherlands
SO Proceedings of the National Academy of Sciences of the United States of America, (1995) Vol. 92, No. 25, pp. 11456-11460.
ISSN: 0027-8424.
DT Article
LA English
AB Integration of human immunodeficiency virus (HIV) DNA into the human genome requires the virus-encoded integrase (IN) protein, and therefore the IN protein is a suitable target for antiviral strategies. To find a potent HIV IN ***inhibitor*** , we screened a "synthetic peptide combinatorial library." We identified a hexapeptide with the sequence HCKFWW that ***inhibits*** IN-mediated 3'-processing and integration with an IC-50 of 2 mu-M. The peptide is active on IN proteins from other retroviruses such as HIV-2, feline immunodeficiency virus, and Moloney murine leukemia virus, supporting the notion that a conserved region of IN is targeted. The hexapeptide was also tested in the disintegration reaction. This phosphoryl-transfer reaction can be carried out by the catalytic core of IN alone, and the peptide HCKFWW was found to ***inhibit*** this reaction, suggesting that the hexapeptide acts at or near the catalytic site of IN. Identification of an IN hexapeptide ***inhibitor*** provides proof of concept for the approach, and, moreover, this peptide may be useful for structure-function analysis of IN.

L17 ANSWER 3 OF 6 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1993:206841 BIOSIS
DN PREV199395108066
TI Correction of BA 95027134. Mutational analysis of the integrase protein of human immunodeficiency virus type 2. Correction of publication year from 1922.
AU Van Gent, Dik C.; Groeneger, Antoinette A. M. Oude; ***Plasterk,*** *** Ronald H. A. (1)***
CS (1) Neth. Cancer Inst., Div. Mol. Biol., Plesmanlaan 121, 1066 CX Amsterdam Netherlands Antilles
SO Proceedings of the National Academy of Sciences of the United States of America, (1992) Vol. 89, No. 20, pp. 9598-9602.
ISSN: 0027-8424.
DT Article; Errata
LA English
AB Purified integrase protein (IN) can nick linear viral DNA at a specific site near the ends and integrate nicked viral DNA into target DNA. We have made a series of 43 site-directed point mutants of human immunodeficiency virus type 2 IN and assayed purified mutant proteins for the following activities: site-specific cleavage

of viral DNA (donor cut), integration (strand transfer), and disintegration. In general, the different activities were similarly affected by the mutations. We found three mutations that (almost) totally abolished IN function: Asp-64 fwdarw Val, Asp-116 fwdarw Ile, and Glu-152 fwdarw Leu, whereas 25 mutations did not affect IN function. A few mutations affected the different activities differentially. Near the amino terminus a zinc finger-like sequence motif His-Xaa-3-His-Xaa-20-30-Cys-Xaa-2-Cys is present in all retroviral IN proteins. Two mutations in this region (His-12 fwdarw Leu and Cys-40 fwdarw Ser) strongly ***inhibited*** donor cut but had less effect on strand transfer. The central region of IN is most highly conserved between retroviral INs. Three mutants in this region (Asn-117 dag -ALPHA-R Ile, Asn-120 fwdarw Leu, and Lys-159 fwdarw Val) were ***inhibited*** in strand transfer but were ***inhibited*** less strongly in donor cut. Mutation of Asn-120 (to glycine, leucine, or glutamate) resulted in changes in integration-site preference, suggesting that Asn-120 is involved in interactions with target DNA. We did not find a mutant in which one activity was lost and the others were unaffected, supporting the notion that IN has only one active site for the catalysis of donor cut and strand transfer.

L17 ANSWER 4 OF 6 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1993:50832 BIOSIS
DN PREV199395027134
TI Mutational analysis of the integrase protein of human immunodeficiency virus type 2.
AU Van Gent, Dik C.; Groeneger, Antoinette A. M. Oude; ***Plasterk,*** *** Ronald H. A. (1)***
CS (1) Neth. Cancer Inst., Div. Mol. Biol., Plesmanlaan 121, 1066 CX Amsterdam Netherlands Antilles
SO Proceedings of the National Academy of Sciences of the United States of America, (1922) Vol. 89, No. 20, pp. 9598-9602.
ISSN: 0027-8424.
DT Article; Errata
LA English
AB Purified integrase protein (IN) can nick linear viral DNA at a specific site near the ends and integrate nicked viral DNA into target DNA. We have made a series of 43 site-directed point mutants of human immunodeficiency virus type 2 IN and assayed purified mutant proteins for the following activities: site-specific cleavage of viral DNA (donor cut), integration (strand transfer), and disintegration. In general, the different activities were similarly affected by the mutations. We found three mutations that (almost) totally abolished IN function: Asp-64 fwdarw Val, Asp-116 fwdarw

Ile, and Glu-152 fwdarw Leu, whereas 25 mutations did not affect IN function. A few mutations affected the different activities differentially. Near the amino terminus a zinc finger-like sequence motif His-Xaa-3-His-Xaa-20-30-Cys-Xaa-2-Cys is present in all retroviral IN proteins. Two mutations in this region (His-12 fwdarw Leu and Cys-40 fwdarw Ser) strongly ***inhibited*** donor cut but had less effect on strand transfer. The central region of IN is most highly conserved between retroviral INs. Three mutants in this region (Asn-117 dag -ALPHA-R Ile, Asn-120 fwdarw Leu, and Lys-159 fwdarw Val) were ***inhibited*** in strand transfer but were ***inhibited*** less strongly in donor cut. Mutation of Asn-120 (to glycine, leucine, or glutamate) resulted in changes in integration-site preference, suggesting that Asn-120 is involved in interactions with target DNA. We did not find a mutant in which one activity was lost and the others were unaffected, supporting the notion that IN has only one active site for the catalysis of donor cut and strand transfer.

L17 ANSWER 5 OF 6 CAPLUS COPYRIGHT 1998 ACS
AN 1998:744978 CAPLUS
DN 130:2014
TI Methods of screening compounds useful for prevention of infection or ***pathogenicity***
IN Ausubel, Frederick M.; Rahme, Lawrence G.; Tan, Man-wah; Ruvkun, Gary B.; Mahajan-Miklos, Shalina; Broeks, Annegien; ***Plasterk,*** *** Ronald H. A.*** ; Jander, Georg; Heard, Jacqueline
PA The General Hospital Corp., USA; The Netherlands Cancer Institute
SO PCT Int. Appl., 87 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9850080	A1	19981112	WO 98-US9150	19980508

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

PRAI US 97-852927 19970508

US 97-962750 19971103

AB Screening procedures are disclosed for identifying compds. useful for ***inhibiting*** infection or ***pathogenicity*** . Methods are also disclosed for identifying ***pathogenic*** virulence factors.

L17 ANSWER 6 OF 6 CAPLUS COPYRIGHT 1998 ACS
AN 1996:725341 CAPLUS
DN 126:14746
TI Peptide ***inhibitors*** of viral integrase and their use in pharmaceuticals
IN Houghten, Richard A.; Weber, Patricia A.; ***Plasterk, Ronald H.*** *** A.*** ; Puras, Lutzke Ramon A.
PA Houghten Pharmaceuticals, Inc., USA
SO U.S., 17 pp.
CODEN: USXXAM
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 5578573	A	19961126	US 95-375911	19950120

OS MARPAT 126:14746
AB The present invention provides viral integrase- ***inhibiting*** peptides having the general structure R1R2-His-Cys-Lys-Phe-Trp-Xaa (R1=H, acyl, alkyl, aralkyl; R2=alkyl, aralkyl; Xaa= amino acid or amino acid analog; the stereochem. of the amino acids or amino acid analogs can be D or L; the amino and carboxy termini of the peptide can be modified). The invention also provides a pharmaceutical compn. comprising a viral integrase- ***inhibiting*** peptide and methods of using a viral integrase- ***inhibiting*** peptide in vitro or in vivo to reduce or ***inhibit*** viral integrase activity in a cell and the infectivity of a virus. A combinatorial hexapeptide library was created and tested for in vitro ***inhibition*** of HIV integrase. Hexapeptides with IC50's of 2-210 .mu.M were identified.

=> e jander georg/au

E1	2	JANDER DONALD R/AU
E2	42	JANDER G/AU
E3	18	--> JANDER GEORG/AU
E4	15	JANDER GERHART/AU
E5	1	JANDER GEROGE/AU
E6	31	JANDER H/AU

E7 1 JANDER H A/AU
E8 3 JANDER H D/AU
E9 110 JANDER H P/AU
E10 1 JANDER HELGA/AU
E11 1 JANDER HILDE D/AU
E12 2 JANDER HILDEGARD D/AU

=> s e2-e3

L18 60 ("JANDER G"/AU OR "JANDER GEORG"/AU)

=> dup rem l18

PROCESSING COMPLETED FOR L18
L19 30 DUP REM L18 (30 DUPLICATES REMOVED)

=> d bib ab 1-

YOU HAVE REQUESTED DATA FROM 30 ANSWERS - CONTINUE? Y/(N):y

L19 ANSWER 1 OF 30 CAPLUS COPYRIGHT 1998 ACS
AN 1998:744978 CAPLUS
DN 130:2014
TI Methods of screening compounds useful for prevention of infection or pathogenicity
IN Ausubel, Frederick M.; Rahme, Lawrence G.; Tan, Man-wah; Ruvkun, Gary B.; Mahajan-Miklos, Shalina; Broeks, Annegien; Plasterk, Ronald H. A.; ***Jander, Georg*** ; Heard, Jacqueline
PA The General Hospital Corp., USA; The Netherlands Cancer Institute
SO PCT Int. Appl., 87 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9850080	A1	19981112	WO 98-US9150	19980508

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK,

ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG
PRAI US 97-852927 19970508
US 97-962750 19971103
AB Screening procedures are disclosed for identifying compds. useful for inhibiting infection or pathogenicity. Methods are also disclosed for identifying pathogenic virulence factors.

L19 ANSWER 2 OF 30 CAPLUS COPYRIGHT 1998 ACS
AN 1996:603244 CAPLUS
DN 125:242717
TI Genetic studies on protein folding and protein secretion in Escherichia coli (chaperone proteins)
AU ***Jander, Georg***
CS Harvard Univ., Cambridge, MA, USA
SO (1996) 179 pp. Avail.: From degree-granting institution From: Diss. Abstr. Int., B 1996, 57(5), 3024
DT Dissertation
LA English
AB Unavailable

L19 ANSWER 3 OF 30 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 1
AN 1996:338336 BIOSIS
DN PREV199699060692
TI Biotinylation in vivo as a sensitive indicator of protein secretion and membrane protein insertion.
AU ***Jander, Georg*** ; Cronan., John E., Jr.; Beckwith, Jon (1)
CS (1) Dep. Microbiol. Mol. Genet., Harvard Med. Sch., Boston, MA 02115 USA
SO Journal of Bacteriology, (1996) Vol. 178, No. 11, pp. 3049-3058. ISSN: 0021-9193.
DT Article
LA English
AB Escherichia coli biotin ligase is a cytoplasmic protein which specifically biotinylates the biotin-accepting domains from a variety of organisms. This in vivo biotinylation can be used as a sensitive signal to study protein secretion and membrane protein insertion. When the biotin-accepting domain from the 1.3S subunit of Propionibacterium shermanii transcarboxylase (PSBT) is translationally fused to the periplasmic proteins alkaline phosphatase and maltose-binding protein, there is little or no biotinylation of PSBT in wild-type E. coli. Inhibition of SecA with sodium azide and mutations in SecB, SecD, and SecF, all of which slow down protein secretion, result in biotinylation of PSBT. When PSBT is fused to the E. coli inner membrane protein MalF, it acts as

a topological marker: fusions to cytoplasmic domains of MalF are biotinylated, and fusions to periplasmic domains are generally not biotinylated. If SecA is inhibited by sodium azide or if the SecE in the cell is depleted, then the insertion of the MalF second periplasmic domain is slowed down enough that PSBT fusions in this part of the protein become biotinylated. Compared with other protein fusions that have been used to study protein translocation, PSBT fusions have the advantage that they can be used to study the rate of the insertion process.

L19 ANSWER 4 OF 30 CAPLUS COPYRIGHT 1998 ACS DUPLICATE 2
AN 1995:874562 CAPLUS
DN 123:279148
TI Evidence that the pathway of disulfide bond formation in Escherichia coli involves interactions between the cysteines of DsbB and DsbA
AU Guilhot, Christophe; ***Jander, Georg*** ; Martin, Nancy L.; Beckwith, Jon
CS Dep. Microbiol. Mol. Genetics, Harvard Med. Sch., Boston, MA, 02115, USA
SO Proc. Natl. Acad. Sci. U. S. A. (1995), 92(21), 9895-9
CODEN: PNASA6; ISSN: 0027-8424
DT Journal
LA English
AB Disulfide bond formation is catalyzed in the periplasm of Escherichia coli. This process involves at least two proteins: DsbA and DsbB. Recent evidence suggests that DsbA, a sol. periplasmic protein directly catalyzes disulfide bond formation in proteins, whereas DsbB, an inner membrane protein, is involved in the reoxidn. of DsbA. Here the authors present direct evidence of an interaction between DsbA and DsbB. The authors isolated a dominant neg. mutant of dsbA, dsbAd, where Cys-33 of the DsbA active site is changed to tyrosine. Both DsbAd and DsbA are able to form a mixed disulfide with DsbB, which may be an intermediate in the reoxidn. of DsbA. This complex is more stable with DsbAd. The dominance can be suppressed by increasing the prodn. of DsbB. By using mutants of DsbB in which one or two cysteines have been changed to alanine, the authors show that only Cys-104 is important for complex formation. Therefore, the authors suggest that in vivo, reduced DsbA forms a complex with DsbB in which Cys-30 of DsbA is disulfide-bonded to Cys-104 of DsbB. Cys-104 is rapidly replaced by Cys-33 of DsbA to generate the oxidized form of this protein.

L19 ANSWER 5 OF 30 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1995:2275 BIOSIS
DN PREV199598016575

TI Pathways of disulfide bond formation in proteins in vivo.
AU Bardwell, James (1); Derman, Alan; Belin, Dominique; ***Jander,*** *** Georg*** ; Prinz, Will; Martin, Nancy; Beckwith, Jon
CS (1) Inst. Biophysik und Physikalische Biochem., Univ. Regensburg, D-93040 Regensburg Germany
SO Torriani-Gorini, A. [Editor]; Yagil, E. [Editor]; Silver, S. [Editor]. (1994) pp. 270-275. Phosphate in microorganisms: Cellular and molecular biology.
Publisher: American Society for Microbiology (ASM) Books Division, 1325 Massachusetts Ave. NW, Washington, DC 20005-4171, USA.
Meeting Info.: Pho-93 Symposium Woods Hole, Massachusetts, USA September 12-17, 1993
ISBN: 1-55581-080-2.
DT Book; Conference
LA English

L19 ANSWER 6 OF 30 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 3
AN 1995:40521 BIOSIS
DN PREV199598054821
TI Two cysteines in each periplasmic domain of the membrane protein DsbB are required for its function in protein disulfide bond formation.
AU ***Jander, Georg*** ; Martin, Nancy L.; Beckwith, Jon
CS Dep. Microbiol., Harvard Med. Sch., 200 Longwood Ave., Boston, MA 02115 USA
SO EMBO (European Molecular Biology Organization) Journal, (1994) Vol. 13, No. 21, pp. 5121-5127.
ISSN: 0261-4189.
DT Article
LA English
AB DsbB is a protein component of the pathway that leads to disulfide bond formation in periplasmic proteins of Escherichia coli. Previous studies have led to the hypothesis that DsbB oxidizes the periplasmic protein DsbA, which in turn oxidizes the cysteines in other periplasmic proteins to make disulfide bonds. Gene fusion approaches were used to show that (i) DsbB is a membrane protein which spans the membrane four times and (ii) both the N- and C-termini of the protein are in the cytoplasm. Mutational analysis shows that of the six cysteines in DsbB, four are necessary for proper DsbB function in vivo. Each of the periplasmic domains of the protein has two essential cysteines. The two cysteines in the first periplasmic domain are in a Cys-X-Y-Cys configuration that is characteristic of the active site of other proteins involved in disulfide bond formation, including DsbA and protein disulfide isomerase.

L19 ANSWER 7 OF 30 CAPLUS COPYRIGHT 1998 ACS
AN 1995:650040 CAPLUS
DN 123:79063
TI Pathways of disulfide bond formation in proteins in vivo
AU Bardwell, James; Derman, Alan; Belin, Dominique; ***Jander,*** *** Georg*** ; Prinz, Will; Martin, Nancy; Beckwith, Jon
CS Institut fur Biophysik und Physikalische Biochemie, Universitat Regensburg, Regensburg, D-93040, Germany
SO Phosphate Microorg. (1994), 270-5. Editor(s): Torriani-Gorini, Annamaria; Yagil, Ezra; Silver, Simon. Publisher: Am. Soc. Microbiol., Washington, D. C.
CODEN: 61LVAV
DT Conference; General Review
LA English
AB A review with 25 refs.

L19 ANSWER 8 OF 30 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 4
AN 1993:211672 BIOSIS
DN PREV199395112897
TI A pathway for disulfide bond formation in vivo.
AU Bardwell, James C. A.; Lee, Jie-Oh; ***Jander, Georg*** ; Martin, Nancy; Belin, Dominique; Beckwtih, Jon (1)
CS (1) Dep. Microbiol. Molecular Genetics, Harvard Med. Sch., Boston, MA 02115 USA
SO Proceedings of the National Academy of Sciences of the United States of America, (1993) Vol. 90, No. 3, pp. 1038-1042.
ISSN: 0027-8424.
DT Article
LA English
AB Protein disulfide bond formation in Escherichia coli requires the periplasmic protein DsbA. We describe here mutations in the gene for a second protein, DsbB, which is also necessary for disulfide bond formation. Evidence suggests that DsbB may act by reoxidizing DsbA, thereby regenerating its ability to donate its disulfide bond to target proteins. We propose that DsbB, an integral membrane protein, may be involved in transducing redox potential across the cytoplasmic membrane.

L19 ANSWER 9 OF 30 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 5
AN 1993:360928 BIOSIS
DN PREV199345044353
TI Gene fusion approaches to membrane protein topology.
AU Boyd, Dana; Traxler, Beth; ***Jander, Georg*** ; Prinz, Will; Beckwith, Jon

CS Dep. Microbiol. Mol. Genet., Harvard Med. Sch., Boston, MA 02115 USA
SO Reuss, L. [Editor]; Russell, J. M., Jr. [Editor]; Jennings, M. L. [Editor]. Society of General Physiologists Series, (1993) Vol. 48, pp. 23-37. Society of General Physiologists Series; Molecular biology and function of carrier proteins.
Publisher: Rockefeller University Press 1230 York Avenue, New York, New York 10021, USA.
Meeting Info.: 46th Annual Symposium of the Society of General Physiologists Woods Hole, Massachusetts, USA September 10-13, 1992
ISSN: 0094-7733. ISBN: 0-87470-053-1.
DT Article
LA English

L19 ANSWER 10 OF 30 EMBASE COPYRIGHT 1998 ELSEVIER SCI. B.V.
AN 93137979 EMBASE
TI Gene fusion approaches to membrane protein topology.
AU Boyd D.; Traxler B.; ***Jander G.*** ; Prinz W.; Beckwith J.
CS Department of Microbiology, Harvard Medical School, Boston, MA 02115, United States
SO J. GEN. PHYSIOL., (1993) 101/46TH ANN. SYMP. (23-37).
ISSN: 0022-1295 CODEN: JGPLAD
CY United States
DT Journal
FS 004 Microbiology
029 Clinical Biochemistry
LA English

L19 ANSWER 11 OF 30 CAPLUS COPYRIGHT 1998 ACS
AN 1993:404459 CAPLUS
DN 119:4459
TI A regulatory cascade controls virulence in Vibrio cholerae
AU DiRita, Victor J.; Parsot, Claude; ***Jander, Georg*** ; Mekalanos, John J.
CS Harvard Med. Sch., Boston, MA, 02115, USA
SO Microb. Adhes. Invasion, [Proc. Symp.] (1992), Meeting Date 1990, 77-83. Editor(s): Hook, Magnus; Switalski, Lech. Publisher: Springer, New York, N. Y.
CODEN: 58SJAY
DT Conference; General Review
LA English
AB A review with 10 refs. summarizing the control of toxR transcription by the heat shock response and the cloning of toxT, a new regulatory gene of V. cholerae.

L19 ANSWER 12 OF 30 BIOSIS COPYRIGHT 1998 BIOSIS

AN 1993:112096 BIOSIS
DN PREV199344054496
TI Analysis of membrane protein topology by gene fusion.
AU Boyd, Dana; Traxler, Beth; ***Jander, Georg*** ; Lee, Cathy; Beckwith, Jon
CS Dep. Microbiol. Mol. Genet., Harvard Med. Sch., Boston, Mass. USA
SO Journal of General Physiology, (1992) Vol. 100, No. 6, pp. 2A-3A. Meeting Info.: Forty-sixth Annual Meeting of the Society of General Physiologists Woods Hole, Massachusetts, USA September 10-13, 1992 ISSN: 0022-1295.
DT Conference
LA English

L19 ANSWER 13 OF 30 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 6
AN 1991:386475 BIOSIS
DN BA92:63790
TI REGULATORY CASCADE CONTROLS VIRULENCE IN VIBRIO-CHOLERAE.
AU DIRITA V J; PARSOT C; ***JANDER G*** ; MEKALANOS J J
CS UNIT LAB. ANIMAL MED., DEP. MICROBIOL. IMMUNOL., UNIV. MICHIGAN MED.
SCH., ANN ARBOR, MICH. 48109.
SO PROC NATL ACAD SCI U S A, (1991) 88 (12), 5403-5407. CODEN: PNASA6. ISSN: 0027-8424.
FS BA; OLD
LA English
AB Expression of more than 17 virulence genes in Vibrio cholerae is under the coordinte control of the ToxR protein. ToxR is a transmembrane protein that binds to and activates the promoter of the operon encoding cholera toxin. As yet, the ability of ToxR to activate directly other genes in this regulon has not been demonstrated. We have cloned a gene called toxT from V. cholerae 569B; the toxT gene product, like ToxR, can activate the ctx promoter in Escherichia coli. In addition, expression of other genes identified as members of the ToxR regulon (tcpA, tcpI, aldA, and tagA) can be activated in E. coli by the toxT gene product but not by ToxR. When expressed from a constitutive promoter, the toxT gene product partially suppresses the ToxR- phenotype of a toxR deletion mutant of V. cholerae. The level of toxT mRNA is greatly reduced in a toxR mutant of V. cholerae. In addition, growth conditions under which the ToxR regulon is not expressed also repress the synthesis of toxT mRNA. These results suggest that ToxR controls transcription of toxT, whose product in turn is directly responsible for activation of several virulence genes under ToxR control.

L19 ANSWER 14 OF 30 CAPLUS COPYRIGHT 1998 ACS
AN 1990:410728 CAPLUS
DN 113:10728
TI A hundred years of technical ceramics - 42 years of oxide semiconductor components
AU ***Jander, G.***
CS Ger. Dem. Rep.
SO Hermsdorfer Tech. Mitt. (1990), 30(77), 2450-1
CODEN: HTMTAN; ISSN: 0439-0377
DT Journal; General Review
LA German
AB A review with no refs.

L19 ANSWER 15 OF 30 CAPLUS COPYRIGHT 1998 ACS
AN 1988:569543 CAPLUS
DN 109:169543
TI Concise Textbook of Inorganic and General Chemistry (Kurzes Lehrbuch der Anorganischen und Allgemeinen Chemie)
AU ***Jander, G.*** ; Spandau, H.
CS Fed. Rep. Ger.
SO (1987) Publisher: (Springer-Verlag, Berlin, Fed. Rep. Ger.), 337 pp. DM 49.50.
DT Book
LA German
AB Unavailable

L19 ANSWER 16 OF 30 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 7
AN 1987:296292 BIOSIS
DN BA84:26324
TI IDENTIFICATION OF THE SERRATIA-MARCESCENS HEMOLYSIN DETERMINANT BY
CLONING INTO ESCHERICHIA-COLI.
AU BRAUN V; NEUSS B; RUAN Y; SCHIEBEL E; SCHOEFFLER H; ***JANDER G***
CS MIKROBIOL. II, UNIV. TUEBINGEN, D-7400 TUEBINGEN, FRG.
SO J BACTERIOL, (1987) 169 (5), 2113-2120.
CODEN: JOBAAY. ISSN: 0021-9193.
FS BA; OLD
LA English
AB A cosmid bank of Serratia marcescens was established from which DNA fragments were cloned into the plasmid pBR322, which conferred the chromosomally encoded hemolytic activity to Escherichia coli K-12. By transposon mutagenesis with Tn1000 and Tn5 IS50L::phoA (TnphoA), the coding region was assigned to a DNA fragment, designated hly, comprising approximately 7 kilobases. Two proteins with molecular weights of 61,000 (61K protein) and 160,000 (160K protein) were

expressed by the pBR322 derivatives and by a plasmid which contained the hly genes under the control of a phage T7 promoter and the T7 RNA polymerase. When strongly overexpressed the 160K protein was released by E. coli cells into the extracellular medium concomitant with hemolytic activity. The genes encoding the 61K and the 160K proteins were transcribed in the same direction. Mutants expressing a 160K protein truncated at the carboxy-terminal end were partially hemolytic. Homolysis was progressively inhibited by saccharides with increasing molecular weights from maltotriose (Mr 504) to maltoheptaose (Mr 1,152) and was totally abolished by dextran 4 (Mr 4,000). This result and the observed influx of [14C]sucrose into erythrocytes in the presence of hemolytic E. coli transformants under osmotically protective conditions suggest the formation of defined transmembrane channels by the hemolysin.

L19 ANSWER 17 OF 30 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1988:185500 BIOSIS
DN BR34:88687
TI ON THE REARING AND KEEPING OF THE MONGOLIAN ROCK SPARROW PETRONIA-PETRONIA-BREVIROSTIS.
AU FIEBIG J; ***JANDER G***
CS MUSEUM FUER NATURKUNDE, INVALIDENSTR. 43, DDR-1040 BERLIN.
SO Gefiederte Welt, (1987) 111 (11), 299-301.
CODEN: GEWEBF. ISSN: 0016-5816.
FS BR; OLD
LA German

L19 ANSWER 18 OF 30 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1989:200379 BIOSIS
DN BA87:101283
TI ON JUVENILE DEVELOPMENT AND BEHAVIOR OF MONGOLIAN ROCK SPARROWS
PETRONIA-PETRONIA-BREVIROSTRIS.
AU FIEBIG J; ***JANDER G***
CS MUS. NATURKUNDE, INVALIDENSTRASSE 43, BERLIN, DDR-1040.
SO MITT ZOOL MUS BERL, (1987) 63 (SUPPL), 123-136.
CODEN: MTZMAK. ISSN: 0373-8493.
FS BA; OLD
LA German
AB The paper describes the juvenile development and the behaviour of hand-raised rock sparrows (Petronia p. brevirostris) and presents information on mating behaviour and vocal repertoire. Furthermore the development of body weight, the growth of feathers and the beginning of moult is treated and measurements and weight of 3 adult birds and of eggs are given. The song of female Rock Sparrow is

recorded for the first time. The authors describe how the typical throat spot becomes visible.

L19 ANSWER 19 OF 30 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1987:51175 BIOSIS
DN BR32:21396
TI AUTUMN OBSERVATION OF THE SPANISH SPARROW PASSER-HISPANIOLENSIS IN
BULGARIA.
AU DITTBERNER H; ***JANDER G***
CS KARSTR. 1, BERGEN, DDR-2330.
SO Beitr. Vogelkd., (1986) 32 (3), 187-189.
CODEN: BEVOAI. ISSN: 0005-8211.
FS BR; OLD
LA German

L19 ANSWER 20 OF 30 CAPLUS COPYRIGHT 1998 ACS
AN 1983:190686 CAPLUS
DN 98:190686
TI Introduction to a Inorganic-Chemical Laboratory Course (Including Quantitative Analysis). 12th Ed (Einfuehrung in das Anorganisch-Chemische Praktikum (Einschliesslich der Quantitativen Analyse)
AU ***Jander, G.*** ; Blasius, E.
CS Ger. Dem. Rep.
SO (1983) Publisher: (S. Hirzel Verlag, Leipzig, Ger. Dem. Rep.), 484 pp. mark 32.
DT Book
LA German
AB Unavailable

L19 ANSWER 21 OF 30 CAPLUS COPYRIGHT 1998 ACS
AN 1982:607212 CAPLUS
DN 97:207212
TI Textbook of Analytical and Preparative Inorganic Chemistry with the Exception of Quantitative Analysis. 12th Ed (Lehrbuch der Analytischen und Praeparativen Anorganischen Chemie mit Ausnahme der Quantitativen Analyse)
AU ***Jander, G.*** ; Blasius, E.
CS Ger. Dem. Rep.
SO (1982) Publisher: (S. Hirzel, Leipzig, Ger. Dem. Rep.), 548 pp. mark 18.50.
DT Book
LA German
AB Unavailable

L19 ANSWER 22 OF 30 CAPLUS COPYRIGHT 1998 ACS
AN 1980:103766 CAPLUS
DN 92:103766
TI Textbook of Analytical and Preparative Inorganic Chemistry with the Exception of Quantitative Analysis. 11th Ed (Lehrbuch der Analytischen und Praeparativen Anorganischen Chemie mit Ausnahme der Quantitativen Analyse)
AU ***Jander, G.*** ; Blasius, E.
CS Ger. Dem. Rep.
SO (1979) Publisher: (M. Hirzel, Leipzig, Ger. Dem. Rep.), 556 pp. mark 32.
DT Book
LA German
AB Unavailable

L19 ANSWER 23 OF 30 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1980:174625 BIOSIS
DN BA69:49621
TI STUDIES ON THE SETTLEMENT DENSITY OF BREEDING BIRDS BERLIN CEMETERIES 1972 EAST GERMANY.
AU DOBBERKAU T; ***JANDER G*** ; OTTO W
CS GAUDYSTR. 15, DDR 1058 BERLIN, E. GER.
SO BEITR VOGELKD, (1979) 25 (3-4), 129-166.
CODEN: BEVOAI. ISSN: 0005-8211.
FS BA; OLD
LA German
AB In East Berlin there are 102 cemeteries with a total area of 480 ha. Areas situated advantageously from a traffic standpoint were studied. A table of the breeding birds was made indicating the number of breeding pairs and the relative dominance in the area. Six breeding bird species were listed in descending order of dominance: greenfinch, starling, blackbird, collared turtle-dove, blue titmouse and whitethroat. There has been a displacement of bird species from the center to the peripheral parts of East Berlin. The nesting behaviors of the breeding birds were discussed.

L19 ANSWER 24 OF 30 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1978:10016 BIOSIS
DN BR14:10016
TI FURTHER IDENTIFICATION OF THE BOREAL OWL AEGOLIUS-FUNEREUS FOR THE
DISTRICT OF BRANDENBURG EAST GERMANY.
AU ***JANDER G***
SO Beitr. Vogelkd., (1977) 23 (3), 188.

CODEN: BEVOAI. ISSN: 0005-8211.
FS BR; OLD
LA Unavailable

L19 ANSWER 25 OF 30 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1976:30661 BIOSIS
DN BR12:30661
TI SPANISH SPARROWS PASSER-HISPANIOLENSIS DRINK SEA WATER.
AU ***JANDER G*** ; MOENKE R
SO Beitr. Vogelkd., (1975) 21 (1/2), 157.
CODEN: BEVOAI. ISSN: 0005-8211.
FS BR; OLD
LA Unavailable

L19 ANSWER 26 OF 30 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1976:30654 BIOSIS
DN BR12:30654
TI A JACK SNIPE LYMNOCRYPTES-MINIMUS IN BERLIN IN WINTER.
AU ***JANDER G***
SO Beitr. Vogelkd., (1975) 21 (1/2), 150-151.
CODEN: BEVOAI. ISSN: 0005-8211.
FS BR; OLD
LA Unavailable

L19 ANSWER 27 OF 30 CAPLUS COPYRIGHT 1998 ACS
AN 1975:25377 CAPLUS
DN 82:25377
TI Textbook of Analytical and Preparative Inorganic Chemistry (Lehrbuch der Analytischen und Praparativen Anorganischen Chemie)
AU ***Jander, G.*** ; Blasius, E.
SO (1973) Publisher: (Hirzel Verlag, Stuttgart, Ger.), 541 pp. DM 32.
DT Book
LA Unavailable
AB Unavailable

L19 ANSWER 28 OF 30 CAPLUS COPYRIGHT 1998 ACS
AN 1973:441484 CAPLUS
DN 79:41484
TI Brief Textbook of Inorganic and General Chemistry. 7th ed (Kurzes Lehrbuch der Anorganischen und Allgemeinen Chemie)
AU ***Jander, G.*** ; Spandau, H.
SO (1973) Publisher: (Springer, New York, N. Y.), 314 pp. 14.10 dollars.
DT Book
LA Unavailable

AB Unavailable

L19 ANSWER 29 OF 30 CAPLUS COPYRIGHT 1998 ACS
AN 1973:37659 CAPLUS
DN 78:37659
TI Handbook of Analytical Chemistry, Pt. 3, Vol. 3a: Quantitative Analysis, Aluminum. 2nd ed (Handbuch der Analytischen Chemie, Teil 3, Bd. 3a: Quantitative Analyse, Aluminium)
AU Fresenius, W.; ***Jander, G.*** ; Bensch, H.
SO (1972) Publisher: (Springer, Heidelberg, Ger.), 716 pp. DM 198. Reviewed in: Angew. Chem. 1972, 84(21), 1077(Ger.)
DT Book
LA German
AB Unavailable

L19 ANSWER 30 OF 30 CAPLUS COPYRIGHT 1998 ACS
AN 1973:99928 CAPLUS
DN 78:99928
TI Ignition and combustion of hybrid propellant combinations with hypergolic additives
AU Beckers, Arno; ***Jander, Georg***
CS Tech. Hochsch. Aachen, Aachen, Ger.
SO Erdoel Kohle, Erdgas, Petrochem. Brennst.-Chem. (1972), 25(7), 400-1 CODEN: EKVBAK
DT Journal
LA German
AB A polyethylene-HNO3 hybrid propellant with a hypergolic additive to achieve ignition and reignition conditions in a hybrid-rocket combustion chamber was studied. Ignition platelets 2-3 mm thick prepd. from a 50:50 p-toluidine-p-aminophenol mixt. gave good results. Optimum conditions for rapid ignition and pressure buildup in relation to geometry, chamber pressure (mass flow), and the injection system were investigated.

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E1 32 HEARD J T JR/AU
E2 27 HEARD J W/AU
E3 10 --> HEARD JACQUELINE/AU
E4 1 HEARD JAMES G/AU
E5 2 HEARD JANE E/AU
E6 1 HEARD JAYNE M/AU
E7 81 HEARD JEAN MICHEL/AU
E8 2 HEARD JOHN F/AU
E9 9 HEARD JOHN T JR/AU

E10 1 HEARD JOHN WILLIAM/AU
E11 1 HEARD JONATHAN F/AU
E12 1 HEARD JONATHAN S/AU

=> s e3

L20 10 "HEARD JACQUELINE"/AU

=> dup rem l20

PROCESSING COMPLETED FOR L20
L21 7 DUP REM L20 (3 DUPLICATES REMOVED)

=> d bib ab 1-

YOU HAVE REQUESTED DATA FROM 7 ANSWERS - CONTINUE? Y/(N):y

L21 ANSWER 1 OF 7 CAPLUS COPYRIGHT 1998 ACS
AN 1998:744978 CAPLUS
DN 130:2014
TI Methods of screening compounds useful for prevention of infection or pathogenicity
IN Ausubel, Frederick M.; Rahme, Lawrence G.; Tan, Man-wah; Ruvkun, Gary B.; Mahajan-Miklos, Shalina; Broeks, Annegien; Plasterk, Ronald H. A.; Jander, Georg; ***Heard, Jacqueline***
PA The General Hospital Corp., USA; The Netherlands Cancer Institute
SO PCT Int. Appl., 87 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9850080	A1	19981112	WO 98-US9150	19980508

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

PRAI US 97-852927 19970508

US 97-962750 19971103

AB Screening procedures are disclosed for identifying compds. useful for inhibiting infection or pathogenicity. Methods are also disclosed for identifying pathogenic virulence factors.

L21 ANSWER 2 OF 7 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 1

AN 1997:345302 BIOSIS

DN PREV199799644505

TI Evolutionary diversity of symbiotically induced nodule MADS box genes: Characterization of nmhC5, a member of a novel subfamily.

AU ***Heard, Jacqueline*** ; Caspi, Michal; Dunn, Kathleen (1)

CS (1) Dep. Biol., Boston Coll., Chestnut Hill, MA 02167 USA

SO Molecular Plant-Microbe Interactions, (1997) Vol. 10, No. 5, pp. 665-676.
ISSN: 0894-0282.

DT Article

LA English

AB Unique organs called nodules form on legume roots in response to intracellular infection by soil bacteria in the genus Rhizobium. This study describes a new MADS box gene, nmhC5, which along with nmh7 (J. Heard and K. Dunn, Proc. Natl. Acad. Sci. USA 92:5273-5277, 1995), is expressed in alfalfa (Medicago sativa) root nodules. Together, these genes represent the first putative transcription factors identified in nodules. Expression in a rootderived structure supports the growing sentiment that MADS box proteins have diverse roles in plant development. Comparison of the putative translation product of nmhC5 with those of other reported members of the MADS box family suggests that the overall structure of nmhC5 is conserved. Evolutionary analysis among the MADS box family showed that nmhC5 is orthologous to a root-expressed clone in Arabidopsis thaliana, agl17, and that nmh7 is similar to the floral subfamily with AP3 (DefA)/PI (Glo). Consistent with a prediction of homodimer formation, NMHC5 was shown to bind a CArG consensus sequence in vitro. In contrast, NMH7, which shows structural similarity to MADS box proteins that form heterodimers, did not bind the CArG element in an in vitro DNA-binding assay, suggesting the existence of an unknown dimer partner. The root-derived MADS box genes constitute a novel subfamily of vegetatively expressed MADS box genes. The evolutionary diversity between nmh7 and nmhC5 could represent an overall mechanistic conservation between plant developmental processes or could mean that nmh7 and nmhC5 make fundamentally different contributions to the development of the nodule.

L21 ANSWER 3 OF 7 CAPLUS COPYRIGHT 1998 ACS

AN 1997:235898 CAPLUS

DN 126:234296
TI Identification and characterization of MADS box genes expressed during alfalfa (Medicago sativa) root nodule development
AU ***Heard, Jacqueline***
CS Boston College, Chestnut Hill, MA, USA
SO (1996) 151 pp. Avail.: Univ. Microfilms Int., Order No. DA9707893 From: Diss. Abstr. Int., B 1997, 57(10), 6077
DT Dissertation
LA English
AB Unavailable

L21 ANSWER 4 OF 7 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 2
AN 1995:361789 BIOSIS
DN PREV199598376089
TI Symbiotic induction of a MADS-box gene during development of alfalfa root nodules.
AU ***Heard, Jacqueline*** ; Dunn, Kathleen (1)
CS (1) Dep. Biol., Boston Coll., Chestnut Hill, MA 02167 USA
SO Proceedings of the National Academy of Sciences of the United States of America, (1995) Vol. 92, No. 12, pp. 5273-5277. ISSN: 0027-8424.
DT Article
LA English
AB In response to infection by Rhizobium, highly differentiated organs called nodules form on legume roots. Within these organs, the symbiotic association between the host plant and bacteria is established. A putative plant transcription factor, NMH7, has been identified in alfalfa root nodules. nmh7 contains a MADS-box DNA-binding region and shows homology to flower homeotic genes. This gene is a member of a multigene family in alfalfa and was identified on the basis of nucleic acid homology to plant regulatory protein genes (MADS-box-containing genes) from Antirrhinum and Arabidopsis. RNA analysis and in situ hybridization showed that expression of this class of regulatory genes is limited to the infected cells of alfalfa root nodules and is likely to be involved in the signal transduction pathway initiated by the bacterial symbiont, Rhizobium meliloti. The expression of nmh7 in a root-derived organ is unusual for this class of regulatory genes.

L21 ANSWER 5 OF 7 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1995:279163 BIOSIS
DN PREV199598293463
TI Symbiotic induction of MADS-box genes during development of alfalfa root nodules.
AU Dunn, Kathleen; ***Heard, Jacqueline***

CS Dep. Biol., Boston Coll., Chestnut Hill, MA 02167 USA
SO Journal of Cellular Biochemistry Supplement, (1995) Vol. 0, No. 21A, pp. 491.
Meeting Info.: Keystone Symposium on Signal Transduction in Plants Hilton Head Island, South Carolina, USA March 29-April 4, 1995
ISSN: 0733-1959.
DT Conference
LA English

L21 ANSWER 6 OF 7 BIOSIS COPYRIGHT 1998 BIOSIS DUPLICATE 3
AN 1995:298591 BIOSIS
DN PREV199598312891
TI Characterization of a nodule-enhanced glutamine synthetase from alfalfa: Nucleotide sequence, in situ localization, and transcript analysis.
AU Temple, Stephen J. (1); ***Heard, Jacqueline*** ; Ganter, Geoffrey; Dunn, Kathleen; Sengupta-Gopalan, Champa (1)
CS (1) Plant Genetic Eng. Lab., Dep. Agron. Hortic., New Mexico State Univ., Las Cruces, NM 88003 USA
SO Molecular Plant-Microbe Interactions, (1995) Vol. 8, No. 2, pp. 218-227.
ISSN: 0894-0282.
DT Article
LA English
AB We have characterized two glutamine synthetase (GS) cDNA clones (pGS13 and pGS100) representing mRNA from root nodules of alfalfa. pGS13 is a full-length version of a previously isolated partial cDNA from an alfalfa nodule cDNA library, while pGS100 was previously isolated from an alfalfa suspension culture cDNA library. Using the 3' untranslated region of the two cDNAs as gene-specific probes, we have shown that the GS genes represented by pGS100 and pGS13 are expressed in all organs tested, although at varying levels. pGS13, however, represents the nodule-enhanced GS gene class. Genomic Southern blot analysis using gene-specific probes shows multiple hybridizing bands, in each case suggesting multiple genes and/or alleles for each class of cytoplasmic GS genes. In situ hybridization of alfalfa nodule sections with gene-specific antisense RNA probes has shown that the nodule-enhanced GS genes are induced in the invasion zone and that their expression is limited to the symbiotic zone, while the GS genes represented by pGSIOO are induced in the early symbiotic zone and are expressed throughout the symbiotic and senescent zones. Transcripts for both sets of GS genes are localized in the infected cells and based on the spatial expression pattern it would appear that the two gene classes are induced independently of the onset of nitrogen fixation.

L21 ANSWER 7 OF 7 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1994:422942 BIOSIS
DN PREV199497435942
TI Expression of a homeotic gene during alfalfa Medicago sativa root nodule development.
AU ***Heard, Jacqueline*** ; Dunn, Kathleen
CS Boston Coll., Dep. Biol., Chestnut Hill, MA 02167 USA
SO Palacios, R. [Editor]; Mora, J. [Editor]; Newton, W. E. [Editor]. Current Plant Science and Biotechnology in Agriculture, (1993) Vol. 17, pp. 344. Current Plant Science and Biotechnology in Agriculture; New horizons in nitrogen fixation.
Publisher: Kluwer Academic Publishers PO Box 989, 3300 AZ Dordrecht, Netherlands.
Meeting Info.: 9th International Congress on Nitrogen Fixation Cancun, Mexico December 6-12, 1992
ISSN: 0924-1949. ISBN: 0-7923-2207-X.
DT Book; Conference
LA English

=> s pathogen? and inhibitor?

3 FILES SEARCHED...
6 FILES SEARCHED...
L22 52825 PATHOGEN? AND INHIBITOR?

=> s l22 and eukaryot?

L23 356 L22 AND EUKARYOT?

=> dup rem l23

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L24 152 DUP REM L23 (204 DUPLICATES REMOVED)

=> s l22 and plant? and vertebrat?

1 FILES SEARCHED...
2 FILES SEARCHED...
L25 311 L22 AND PLANT? AND VERTEBRAT?

=> dup rem l25

PROCESSING COMPLETED FOR L25
L26 302 DUP REM L25 (9 DUPLICATES REMOVED)

=> s l26 and invertebrate?

L27 20 L26 AND INVERTEBRATE?

=> d bib ab 1-
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YOU HAVE REQUESTED DATA FROM 20 ANSWERS - CONTINUE? Y/(N):y

L27 ANSWER 1 OF 20 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1998:514777 BIOSIS
DN PREV199800514777
TI Effect of a killer toxin of Pichia anomala to Pneumocystis. Perspectives in the control of pneumocystosis.
AU Seguy, Nathalie (1); Polonelli, Luciano; Dei-Cas, Eduardo; Cailliez, Jean-Charles
CS (1) Dep. Microbiol. Ecosystemes, Inst. Pasteur Lille, 1 rue Prof. Calmette, BP 245, 59019 Lille France
SO FEMS Immunology and Medical Microbiology, (Sept., 1998) Vol. 22, No. 1-2, pp. 145-149.
ISSN: 0928-8244.
DT Article
LA English
AB Despite the development of drugs in the prophylaxis of pneumocystosis, Pneumocystis carinii remains a major opportunistic microorganism in immunosuppressed individuals, especially in human immunodeficiency virus-infected patients. Since side effects were frequently observed after administration of trimethoprim-sulfamethoxazole or pentamidine, the drugs which are mainly used in treating human P. carinii pneumonia (PCP), new therapeutic strategies should be developed. Over the last years, the ***inhibitory*** effect of a Pichia anomala killer toxin (PaKT), a molecule with a wide spectrum of antimicrobial activity, was characterized on P. carinii. The susceptibility of mouse and rat-derived Pneumocystis to PaKT has been demonstrated by in vitro attachment tests and in vivo infectivity assays. Nevertheless, PaKT is strongly antigenic, toxic and could not be used directly as a therapeutic agent. Then, a new strategy using killer toxin-like anti-idiotypic antibodies (KT-antiIds) mimicking the fungal toxin activity has been developed. Different KT-antiIds were obtained by idiotypic immunization with a monoclonal antibody (mabKT4). This mabKT4 neutralized the killer properties of the PaKT. KT-antiIds were produced by immunization against the variable domain (idiotype) of mAbKT4 (internal image of the killer toxin receptor), or they

were obtained directly from vaginal fluid of patients affected by recurrent vaginal candidiosis. In this last case, such natural KT-antiIds were immunopurified by affinity-chromatography with mAbKT4 and their anti-P. carinii activity was then evaluated. Our results showed that both the in vitro attachment of rat-derived parasites and their infectivity to nude rats were inhibited by the KT-antiIds. With regard to KT-antiIds obtained by immunization, the antimicrobial activity of a monoclonal KT-antiIds (mAbK10) has been evaluated by using a PCP experimental nude rat model treated by mAbK10 administered by aerosol. The pneumocystosis extension was significantly reduced in this model. The monoclonal KT-antiIds were effective against P. carinii in reducing parasite proliferation in lungs of nude rats. Further experiments are in progress to study the in vivo anti-P. carinii activity of KT-antiIds by using recombinant single-chain of the variable fragment of KT-antiIds. Yeast killer toxin-like recombinant molecules could provide the basis for a new therapeutic strategy towards the control of pneumocystosis.

L27 ANSWER 2 OF 20 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1998:132087 BIOSIS
DN PREV199800132087
TI Identification and characterization of an endo/exonuclease in Pneumocystis carinii that is inhibited by dicationic diarylfurans with efficacy against Pneumocystis pneumonia.
AU Hildebrandt, Ellen; Boykin, David W.; Kumar, Arvind; Tidwell, Richard R.; Dykstra, Christine C. (1)
CS (1) Dep. Pathobiol., Coll. Veterinary Med., Auburn Univ., Auburn, AL 36849 USA
SO Journal of Eukaryotic Microbiology, (Jan.-Feb., 1998) Vol. 45, No. 1, pp. 112-121.
ISSN: 1066-5234.
DT Article
LA English
AB Dicationic diarylfurans and dicationic carbazoles are under development as therapeutic agents against opportunistic infections. While their ability to bind to the minor groove of DNA has been established, the complete mechanism of action has not. We demonstrate here that an effective diarylfuran, 2,5-bis(4-(N-isopropylguanyl)phenyl)furan, inhibits an endo/exonuclease activity present in Pneumocystis carinii, Cryptococcus neoformans, Candida albicans, and Saccharomyces cerevisiae. This activity was purified from the particulate fraction of P. carinii. The enzyme requires Mg++ or Mn++, and shows preferences for single-over double stranded DNA and for AT-rich over GC-rich domains. A panel of 12 dicationic diarylfurans and eight dicationic carbazoles, previously

synthesized, were evaluated for inhibition of the purified nuclease and for efficacy against Pneumocystis pneumonia in rats. Among the diarylfurans, potency of nuclease inhibition, in vivo antimicrobial activity, and DNA binding strength were all strongly correlated ($p < 0.001$). These findings suggest that one target for antimicrobial action of the diarylfurans may be a nucleolytic or other event requiring unpairing of DNA strands. Dicationic carbazoles which were strong nuclease ***inhibitors*** all displayed anti-Pneumocystis activity in vivo, but there were also noninhibitory carbazoles with in vivo efficacy.

L27 ANSWER 3 OF 20 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1997:491214 BIOSIS
DN PREV199799790417
TI ***Inhibitory*** effect of human natural yeast killer toxin-like candidacidal antibodies on Pneumocystis carinii.
AU Seguy, Nathalie; Cailliez, Jean-Charles; Delcourt, Philippe; Conti, Stefania; Camus, Daniel; Dei-Cas, Eduardo; Polonelli, Luciano (1)
CS (1) Ist. Microbiol., Faculta Med. Chirurgia, Univ. Studi Parma, Via A. Gramsci 14, 43100 Parma Italy
SO Molecular Medicine (New York), (1997) Vol. 3, No. 8, pp. 544-552. ISSN: 1076-1551.
DT Article
LA English
AB Background: Human natural antibodies have been found that owe their candidacidal action to the mimicry of a yeast killer toxin produced by the yeast Pichia anomala (PaKT). Candidacidal human natural antibodies (KTAb) are elicited by and bind to a Kr receptor (PaKTR) present on the cell surface of infectious PaKT-sensitive microorganisms. Because of the recognized susceptibility of Pneumocystis carinii organisms to PaKT upon the occurrence of specific PaKTR, we examined whether human natural KTAb could also bind to and inhibit P. carinii. Materials and Methods: Immunoaffinity-purified KTAb from the vaginal fluid of patients affected by candidiasis were tested and compared with PaKT for their ability to inhibit rat-derived P. carinii attachment to epithelial lung cells as well as infectivity to nude rats. Immunofluorescence studies were also performed by biotinylated PaKT in competition with human KTAb to establish their specific binding to PaKTR on the surface of rat-derived and human P. carinii organisms. Results: Human natural candidacidal KTAb exerted a strong, specific ***inhibitory*** activity against rat-derived P. carinii organisms that are susceptible to PaKT itself. The antimicrobial activity of human KrAb was abolished by adsorption with a specific PaKT-neutralizing mAb KT4. Immunofluorescence studies of competition

with PaKT showed that human KTAb efficiently bind to the specific PaKTR on the surface of rat-derived and human P. carinii organisms. Conclusions: The results strongly suggest that human KTAb, elicited by a common transphyletic receptor of different ***pathogenic*** microorganisms during infection, may play a role in antibody-mediated cross-immunity and, if properly engineered, as functionally equivalent recombinant antibodies they could exert a therapeutic activity against pneumocystosis in vivo.

L27 ANSWER 4 OF 20 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1997:450084 BIOSIS
DN PREV199799749287
TI Lipid biosynthesis pathways as chemotherapeutic targets in kinetoplastid parasites.
AU Durbina, J. A.
CS Laboratorio de Quimica Biologica, Centro de Bioquimica y Biofisica, Instituto Venezolano de Investigaciones Cientificas, Apartado 21827, Caracas 1020 Venezuela
SO Parasitology, (1997) Vol. 114, No. SUPPL., pp. S91-S99. ISSN: 0031-1820.
DT General Review
LA English
AB ***Inhibitors*** of sterol and phospholipid biosynthesis in kinetoplastid parasites such as Trypanosoma cruzi, the causative agent of Chagas' disease, and different species of Leishmania have potent and selective activity as chemotherapeutic agents in vitro and in vivo. Recent work with the sterol C14-alpha-demethylase ***inhibitor*** D0870, a his triazole derivative, showed that this compound is capable of inducing radical parasitological cure in murine models of both acute and chronic Chagas' disease. Other ***inhibitors*** of this type, such as SCH 56592, have also shown curative, rather than suppressive, activity against T. cruzi in these models. Leishmania species have different susceptibilities to sterol biosynthesis ***inhibitors*** , both in vitro and in vivo. Leishmania braziliensis promastigotes, naturally resistant to C14-alpha-demethylase ***inhibitors*** such as ketoconazole and D0870, were susceptible to these drugs when used in combination with the squalene epoxidase ***inhibitor*** terbinafine. ***Inhibitors*** of DELTA-24(25) sterol methyl transferase have been shown to act as potent antiproliferative agents against Trypanosoma cruzi, both in vitro and in vivo. New ***inhibitors*** of this type which show enhanced activity and novel mechanisms of action have been synthesized. Recent work has also demonstrated that this type of enzyme ***inhibitors*** can block sterol biosynthesis and cell proliferation in Pneumocystis carinii, a

fungal ***pathogen*** which had previously been found resistant to other sterol biosynthesis ***inhibitors*** . Ajoene, an antiplatelet compound derived from garlic, was shown to have potent antiproliferative activity against epimastigotes and amastigotes of Trypanosoma cruzi in vitro; this activity was associated with a significant alteration of the phospholipid composition of the cells with no significant effects on the sterol content. In addition, alkyllsophospholipids such as ilmofosine, miltefosine and edelfosine have been shown to block the proliferation of T. cruzi and Leishmania and alter both the phospholipid and sterol composition. These results indicate the potential of lipid biosynthesis ***inhibitors*** as useful therapeutic agents in the treatment of leishmaniasis and Chagas' disease.

L27 ANSWER 5 OF 20 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1997:433144 BIOSIS
DN PREV199799732347
TI Production of thiotropocin by a marine bacterium, Caulobacter sp. and its antimicroalgal activities.
AU Kawano, Yasuhiro (1); Nagawa, Yoshinobu; Nakanishi, Hiroshi; Nakajima, Hirofumi; Matsuo, Masaru; Higashihara, Takanori
CS (1) Natl. Inst. Biosci. Human-Technol., 1-1 Higashi, Tsukuba, Ibaraki 305 Japan
SO Journal of Marine Biotechnology, (1997) Vol. 5, No. 4, pp. 225-229. ISSN: 0941-2905.
DT Article
LA English
AB Marine microalgae were isolated from coastal seawater in Japan to elucidate the mechanisms of interaction between microalgae and marine bacteria. More than 200 strains of marine bacteria have been isolated from unialgal cultures. As a first screening for chemical substances that cause interaction between microalgae and marine bacteria, the antimicrobial activities of isolated marine bacteria were examined. An isolated strain, PK654, showed considerable antimicrobial activity. The PK654 strain was identified as Caulobacter sp. and was shown to produce the antibiotic, thiotropocin. The compound had a strong ***inhibitory*** effect on the growth of the microalgae, Skeletonema costatum and Heterosigma akashiwo, red tide phytoplankton at a concentration of 1 mu-g/ml and caused inhibition of Enterococcus seriolicida, a ***pathogen*** of the Japanese amberjack or yellowtail, Seriola quiqueradiata, at a concentration of 13.3 mu-g/ml.

L27 ANSWER 6 OF 20 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1997:390179 BIOSIS

DN PREV199799689382
TI Human and Saccharomyces cerevisiae dolichol phosphate mannose synthases represent two classes of the enzyme, but both function in Schizosaccharomyces pombe.
AU Colussi, Paul A.; Taron, Christopher H.; Mack, Jamey C.; Orlean, Peter (1)
CS (1) Dep. Biochemistry, Univ. Illinois, 309 Roger Adams Lab., 600 South Mathews Ave., Urbana, IL 61801 USA
SO Proceedings of the National Academy of Sciences of the United States of America, (1997) Vol. 94, No. 15, pp. 7873-7878.
ISSN: 0027-8424.
DT Article
LA English
AB Dolichol phosphate mannose (Dol-P-Man), formed upon transfer of Man from GDPMan to Dol-P, is a mannosyl donor -n pathways leading to N-glycosylation, glycosyl phosphatidylinositol membrane anchoring, and O-mannosylation of protein. Dol-P-Man synthase is an essential protein in Saccharomyces cerevisiae. We have cloned cDNAs encoding human and Schizosaccharomyces pombe proteins that resemble S. cerevisiae Dol-P-Man synthase. Disruption of the gene for the S. pombe Dol-P-Man synthase homolog, dpm1+, is lethal. The known Dol-P-Man synthase sequences can be divided into two classes. One contains the S. cerevisiae, Ustilago maydis, and Trypanosoma brucei enzymes, which have a COOH-terminal hydrophobic domain, and the other contains the human, S. pombe, and Caenorhabditis synthases, which lack a hydrophobic COOH-terminal domain. The two classes of synthase are functionally equivalent, because S. cerevisiae DPM1 and its human counterpart both complement the lethal null mutation in S. pombe dpm1+. The findings that Dol-P-Man synthase is essential in yeast and that the Ustilago and Trypanosoma synthases are in a different class from the human enzyme raise the possibility that Dol-P-Man synthase could be exploited as a target for ***inhibitors*** of ***pathogenic*** eukaryotic microbes.

L27 ANSWER 7 OF 20 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1997:195522 BIOSIS
DN PREV199799494725
TI LY303366 single dose pharmacokinetics and safety in healthy male volunteers.
AU Lucas, R. (1); Desante, K.; Hatcher, B.; Hemingway, J.; Lachno, R.; Brooks, S.; Turik, M.
CS (1) Lilly Res. Centre, Surrey UK
SO Abstracts of the Interscience Conference on Antimicrobial Agents and Chemotherapy, (1996) Vol. 36, No. 0, pp. 108.
Meeting Info.: 36th ICAAC (International Conference of Antimicrobial

Agents and Chemotherapy) New Orleans, Louisiana, USA September 15-18, 1996
DT Conference; Abstract; Conference
LA English

L27 ANSWER 8 OF 20 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1996:421825 BIOSIS
DN PREV199699144181
TI A molecular mechanism for the effect of lithium on development.
AU Klein, Peter S. (1); Melton, Douglas A.
CS (1) Howard Hughes Med. Inst., Dep. Med., Inst. Aging, Univ. Pennsylvania, Philadelphia, PA 19104 USA
SO Proceedings of the National Academy of Sciences of the United States of America, (1996) Vol. 93, No. 16, pp. 8455-8459.
ISSN: 0027-8424.
DT Article
LA English
AB Lithium, one of the most effective drugs for the treatment of bipolar (manic-depressive) disorder, also has dramatic effects on morphogenesis in the early development of numerous organisms. How lithium exerts these diverse effects is unclear, but the favored hypothesis is that lithium acts through inhibition of inositol monophosphatase (IMPase). We show here that complete inhibition of IMPase has no effect on the morphogenesis of Xenopus embryos and present a different hypothesis to explain the broad action of lithium. Our results suggest that lithium acts through inhibition of glycogen synthase kinase-3-beta (GSK-3-beta), which regulates cell fate determination in diverse organisms including Dictyostelium, Drosophila, and Xenopus. Lithium potently inhibits GSK-3-beta activity (K-i = 2 mM), but is not a general ***inhibitor*** of other protein kinases. In support of this hypothesis. lithium phenocopies loss of GSK-3-beta function in Xenopus and Dictyostelium. These observations help explain the effect of lithium on cell-fate determination and could provide insights into the ***pathogenesis*** and treatment of bipolar disorder.

L27 ANSWER 9 OF 20 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1996:321506 BIOSIS
DN PREV199699043862
TI Recent developments and rationale towards new strategies for malarial chemotherapy.
AU Vial, H.
CS Dynamique Mol. Interactions Membranaires, CNRS URA 1856, Univ. Montpellier II, Case 107, Place Eugene-Bataillon, 34095 Montpellier Cedex 5 France

SO Parasite, (1996) Vol. 3, No. 1, pp. 3-23.
ISSN: 1252-607X.
DT Article
LA English
SL English; French
AB The major problem facing world research for new antimalarials lies in encountered difficulties in the search for new promising paths. The past 20 years have witnessed a very impressive increase in our understanding of the biochemistry and molecular biology of malaria parasites, with attention focused on specific parasite molecules that are keys to the parasite life cycle or the induction of its ***pathogenesis*** . Directed pharmacology research has involved the identification and characterization of targets that can be specifically pharmacologically affected, including the replicating machinery of the parasites, various metabolisms such as the purine salvage pathway, and biosynthesis of pyrimidines or phospholipids. Protease ***inhibitors*** (e.g. those degrading haemoglobin), the use of iron chelators or inhibition of heme polymerization, induction of oxidative stress or inhibition of antioxidant enzymes are also investigated. Some pathways have already been validated with current antimalarials, but, due to the development of resistance, complete characterization of the molecular structure of the target should allow attack of these exceptional molecules at novel and distinct sites with new drug concepts. The problem in the quest to develop new antimalarials is the fact that the results are not being materialized, but there is no lack of pharmacological targets.

L27 ANSWER 10 OF 20 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1996:76700 BIOSIS
DN PREV199698648835
TI Genetic transformation of the ***pathogenic*** fungus Wangiella dermatitidis.
AU Peng, M.; Cooper., C. R., Jr.; Szaniszlo, P. J. (1)
CS (1) Dep. Microbiology, Univ. Texas Austin, Austin, TX 78712-1095 USA
SO Applied Microbiology and Biotechnology, (1995) Vol. 44, No. 3-4, pp. 444-450.
ISSN: 0175-7598.
DT Article
LA English
AB Genetic transformation of Wangiella dermatitidis was studied using three plasmid vectors (pAN7-1, pWU44, and pKK5) and both electroporation and polyethyleneglycol-mediated methods. pAN7-1 contains the E. coli hygromycin B (HmB) phosphotransferase (hph) gene. Expression of the hph gene confers resistance to antibiotic

HmB. Selection for resistance, indicative of transformation, resulted in 10-203 HmB-resistant colonies/mu-g pAN7-1 on medium containing 100 mu-g HmB/ml. Strains of W. dermatitidis used in this study have innate sensitivity to HmB at a critical ***inhibitory*** concentration of 20-40 mu-g/ml. Vectors pWU44 and pKK5 contain a URA5 gene from Podospora anserina. A ura5 auxotroph of W. dermatititis was transformed to prototrophy with pWU44 or pKK5 by complementation. Transformation frequencies for these two plasmids were between 17-50 transformants/mu-g vector DNA. Southern blotting analysis and polymerase chain reaction detection of DNA from putative transformants confirmed transformation.

L27 ANSWER 11 OF 20 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1996:20774 BIOSIS
DN PREV199698592909
TI Species-specific microhelix aminoacylation by a eukaryotic ***pathogen*** rRNA synthetase dependent on a single base pair.
AU Quinn, Cheryl L. (1); Tao, Nianjun; Schimmel, Paul
CS (1) Cubist Pharmaceutical Inc., 24 Emily St., Cambridge, MA 02139 USA
SO Biochemistry, (1995) Vol. 34, No. 39, pp. 12489-12495. ISSN: 0006-2960.
DT Article
LA English
AB We report here that tyrosyl-tRNA synthetase from the eukaryotic ***pathogen*** Pneumocystis carinii is a 370 amino acid polypeptide with characteristic elements of a class I aminoacyl-tRNA synthetase and aligns with the prokaryotic tyrosyl-tRNA synthetases in the class-defining active site region, including the tRNA acceptor helix-binding region. The expressed enzyme is a dimer that aminoacylates yeast tRNA but not Escherichia coli tRNA-Tyr. Like most tRNAs, prokaryotic tyrosine tRNAs have a G1 cntdot C72 base pair at the ends of their respective acceptor helices. However, the eukaryote cytoplasmic tyrosine tRNAs have an uncommon C1 cntdot G72 base pair. We show that P. carinii tyrosyl-tRNA synthetase charges a seven base pair hairpin microhelix (microhelix-Tyr) whose sequence is derived from the acceptor stem of yeast cytoplasmic tRNA-Tyr. In contrast, the enzyme does not charge E. coli microhelix-Tyr. Changing the C1 cntdot G72 of yeast microhelix-Tyr to G1 cntdot C72 abolishes charging by the P. carinii tyrosyl-tRNA synthetase. Conversely, we found that E. coli tyrosyl-tRNA synthetase can charge an E. coli microhelux-Tyr and that charging is sensitive to having a G1 cntdot C72 rather than a C1 cntdot G72 base pair. The results demonstrate that the common structural framework of homologous tRNA synthetases has the capacity to coadapt to a transversion in a

critical acceptor helix base pair and that this coadaptation can account for species-selective microhelix aminoacylation. We propose that species-selective acceptor helix recognition can be used as a conceptual basis for species-specific ***inhibitors*** of tRNA synthetases.

L27 ANSWER 12 OF 20 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1994:123373 BIOSIS
DN PREV199497136373
TI Antitrichomonal action of emodin in mice.
AU Wang, Hwang-Huei
CS Div. Gastroenterol., Dep. Intern. Med., Hosp. China Med. Coll., Taichung City 40408 Taiwan
SO Journal of Ethnopharmacology, (1993) Vol. 40, No. 2, pp. 111-116.
ISSN: 0378-8741.
DT Article
LA English
AB Emodin, an active component contained in the root and rhizome of Rheum palmatum L. (Polygonaceae), was found to have an ***inhibitory*** effect on the ***pathogenicity*** of Trichomonas vaginalis in mice. Emodin delayed the development of subcutaneous abscesses due to infection of this parasite. Also, it cures the intravaginal infection of trichomonads through oral administration. In cell cultures, it reduced the cytotoxic effect of this parasite towards mammalian cells. This inhibition was markedly reversed by the coexistence of free radical scavengers, indicating the possible mediation of free radicals.

L27 ANSWER 13 OF 20 BIOSIS COPYRIGHT 1998 BIOSIS
AN 1993:520130 BIOSIS
DN PREV199396133537
TI Dihydrofolate reductase from the ***pathogenic*** fungus Pneumocystis carinii: Catalytic properties and interaction with antifolates.
AU Margosiak, Stephen A.; Appleman, James R.; Santi, Daniel V.; Blakley, Raymond L. (1)
CS (1) Dep. Mol. Pharmacol., St. Jude Children's Res. Hosp., Memphis, TN 38101 USA
SO Archives of Biochemistry and Biophysics, (1993) Vol. 305, No. 2, pp. 499-508.
ISSN: 0003-9861.
DT Article
LA English
AB Dihydrofolate reductase (DHFR) from the fungus Pneumocystis carinii (pcDHFR), a target for antifolate ***inhibitors*** , has been